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BRAIN.

PART I., 1906.

Original Articles and Clinical Cases.

UPON THE ORIENTATION OF POINTS IN SPACE BY THE MUSCULAR, ARTHRODIAL, AND TACTILE SENSES OF THE UPPER LIMBS IN NORMAL INDIVIDUALS AND IN BLIND PERSONS.

(Mesial and Lateral Sagittal Planes.)

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AND

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Introduction.

For purposes of exact localisation and diagnosis of disease the circumstances under which orientation in space becomes part of our normal knowledge requires further investigation. Attention has for many years been drawn to the semi-circular canals, and of course conscious perception of our position in space is very largely effected by the functional activity of those organs; but there is a second and important avenue of information to the brain on the matter of orientation, namely, that of our muscular, arthrodial and tactile senses, which deserves fuller consideration.

It was suggested by Horsley at the meeting of the Neurological Society, June 24, 1905, that for these senses measurement of the error of localisation in space for each of the three planes of space, sagittal, horizontal and frontal (supposing the visual axes to be horizontal), was now scientifically necessary, especially in the investigation of cerebellar disease, and he demonstrated how this could readily be attained by employing[1] a glass plate, graduated in half centimetres, which could be placed in the respective planes, and screened from the patient's sight. (See fig. 1.)

The latter having been directed to place[2] one finger tip against the plate on one side and to touch the corresponding point on the other side of plate with the other finger, any error was easily read off and noted for the two principal axes of that plane.

[1] M. Bloch, [1] in 1884, employed a similar principle for the discrimination between the sense of muscles in action and repose.

[2] Thus in the preliminary work with the plate the person observed had the extra information of the sensations from the "actively" moved limb. In the present communication the subject's finger was "passively" placed for him on the plate.

Preliminary experiments had shown him that the greatest error was in the frontal plane, that this was true for normal as well as abnormal individuals, and further that proximality was the commonest error of antero-posterior localisation.

The further position of the question and use of the method has also been referred to by Horsley [2] in his Boyle Lecture.

The question whether an average constancy of error exists amongst normal individuals concerning their know-

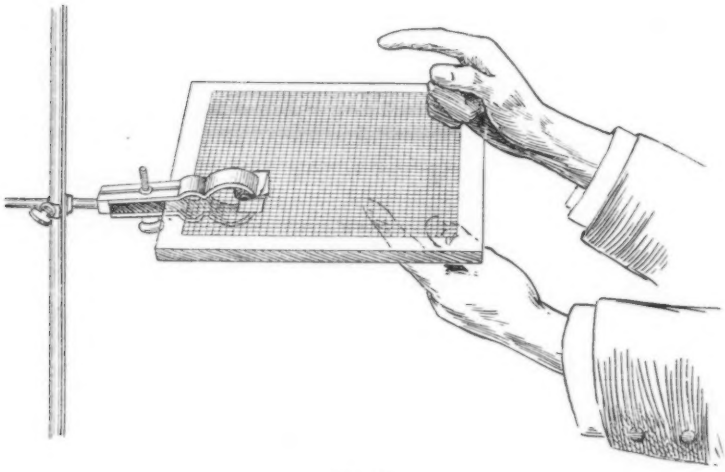


FIG. 1.

ledge of space surrounding them, whether this error is the same for the two halves of the body and of equal value in the different planes of space was then undertaken by Slinger. To this end after a preliminary series of observations in London with the divided plate on normal individuals and a few cases of cerebral lesions, Dr. Slinger proceeded to the Psychological Laboratory at Cambridge under Dr. W. H. R. Rivers, by whose experience in experimental psychology and generous help sundry sources of adventitious error were eliminated.

We wish to take this opportunity of expressing our indebtedness to Dr. Rivers, not only for his criticism and suggestions, but also for his warm interest in the research.

Subsequently, in London, Dr. Slinger made a large number of observations on normal persons and on the blind.

Thus the whole of the observations upon which the following statistical results have been based were made by him.

Method.

In originally devising the plate method the reasons for choosing the mesial sagittal plane as the primary one for investigation were first the bilateral construction of the body which rendered it obvious that the greatest accuracy should *a priori* be expected to accompany determination of points in that plane towards which the upper limbs are in constant synergic movement; secondly, that the selection of this plane obviated any complication due to the structural length of the upper limb and the mechanism of the shoulder and other joints, which anatomical features impose serious conditions on the usefulness and accuracy of the locomotion of the limb; and, thirdly, since (*see* Boyle Lecture) the small shell of space which the upper limbs can explore and investigate has a remarkable indented periphery it appeared advisable to begin with a plane towards which all movements of the limbs were the simplest.

Also in thus selecting the mesial plane all confusion, owing to natural asymmetry and unilaterality was readily avoided by compounding the results obtained by the two hands, and it will be seen presently that the complication of unilaterality is a real source of variation and the method in some degree confirms the views of Guldberg, regarding the circular error of orientation which all animals may exhibit.

The investigation of the errors in planes lateral to the mesial plane was commenced much later, and the results obtained follow on after those obtained from the middle line of the body.

The actual method has been mentioned above, but, like all modes of investigation, there are certain details which

materially affect the accuracy of the plate test and must now be stated.

In the investigation the subject observed must be placed in a chair well supported, and his eyes must be screened before the plate is placed in position in front of him. The investigator must next ascertain the true position of the segments of the limb at the level selected for measurement, and finally the finger (of the subject observed) whose position is to be recorded must be taken by the investigator and placed by him firmly on the plate, the subject observed taking care that it is not subsequently moved until the next observation. The recording finger and limb of the subject observed must be moved freely in the air and brought as nearly as possible vertical to the plate. In this way it will be seen that the method is in fact a direct appreciation of any point in space by means of the muscular and arthrodial senses, or, in other words, locomotion of the limb.[1]

An extremely interesting but serious complication is introduced at once if the individual observed becomes aware of his inaccuracies, and endeavours by mentation to correct the position of the recording finger. It will be obvious that inasmuch as a further error of direction is possible over 360° around the spot actually touched, such mental efforts to better the result usually result in increasing the mistake made.

Of the foregoing points, the most important precaution is that the individual observed should not see the plate until the end of the experiment. The reason is obvious, namely, that the memories of the visual relations of points in space are so numerous and accurate, that they immediately supplant the evidence of such a relatively crude faculty as that of the muscular sense.

The investigator must take care that the visual axes of the person observed are horizontal in this series throughout the experiments. He also must not allow any part of the hand or body of the person observed to touch the plate except the tip of the observed finger, and in counting the

[1] An additional confirmation of this is the fact that practically the same result follows if tactility is not present, *i.e.*, if the observed finger does not touch the plate.

amount of error he should occupy an approximately equal time for each reading, *e.g.* whether easy or difficult, lest an indication should be given to the subject of the degree of error, and so lead him to introduce the complication of mentation above referred to.

Selection of Points in Space to be Investigated.

Among the first questions for consideration was the determination of the points in space at which to make observations for accurate statistical summary. Decision had to be made between arbitrary or anatomical distances, and was settled at once in favour of the latter. A further question arose whether in the exploration of space by the upper limb the memory of movements accomplished is cerebrally arranged in relation to the muscles or to the joints between the segments moved. This matter really includes the all-important generalisation of Dr. Hughlings Jackson, namely, that what are represented in the cerebral[1] cortex are not muscles, but movements; and Beevor and Horsley found in attempting to analyse the movements resulting from cortical excitation that all classification was necessarily expressed in terms of the joints. For these reasons, and being under the necessity of choosing points which should bear the same relation in different individuals to the long axis of the body we selected the elbow, wrist and finger tips as the points to be marked upon the different levels chosen. The lines joining up these points are of course roughly concentrically curved as the fully extended limb is moved around its axis at the shoulder, and form convenient subdivisions of the space under investigation.

The Positions of Levels chosen.

The positions or levels in which the foregoing points are grouped were taken in the following manner as shown in the accompanying figure 2.

Position 1.—Vertically over the head parallel to the longitudinal body axis (" overhead ").

[1] The *cerebral* cortex is alone here referred to.

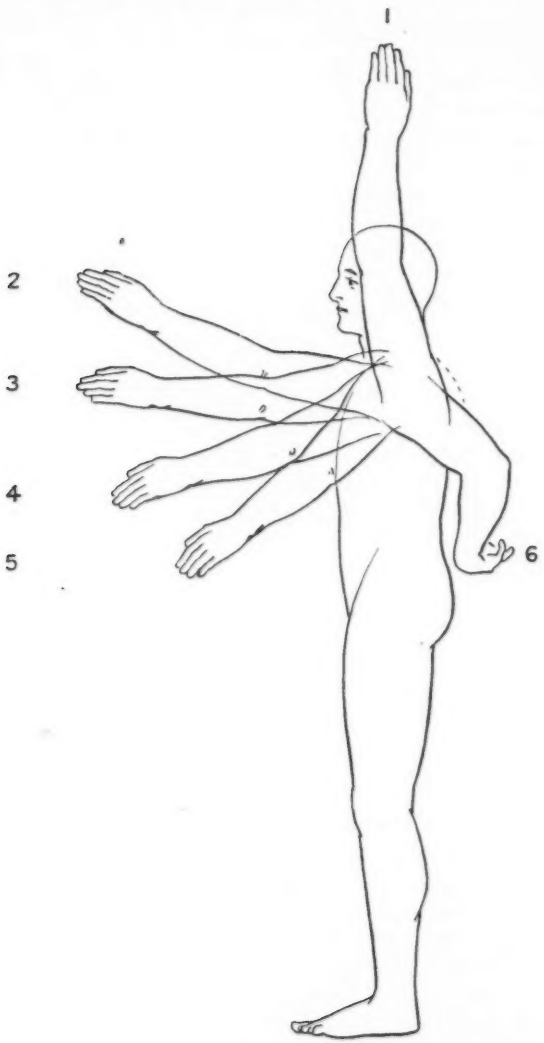


FIG. 2.

The figure is outlined from a number of photographs of the body and upper limbs held successively in the positions enumerated in the text while the finger tips of the two hands met in the mesial sagittal plane.

The actual places in space taken by the different segments of the upper limb are thus correctly determined, and the foreshortening of the parts also shows how the movements of the arm are limited by the joints and tensions of the muscles, tendons, &c. (1) "Overhead," (2) "Visual," (3) "Shoulder," (4) "Ensiform," (5) "Centre of Gravity," (6) "Behind."

Position 2.—A horizontal line passing along the visual axes ("visual").

Position 3.—A horizontal line passing through the centre of the shoulder joint ("shoulder").

Position 4.—A horizontal line passing through the lowest part of the mesosternum ("ensiform").

Position 5.—A horizontal line passing through the centre of gravity of the whole body ("centre of gravity").

Position 6.—On the last-named line, behind the body ("behind").

In the first of these positions the elbow joint often coincides with the level of the top of the head, or is just below it, consequently this nodal point could not in such persons be used in this position.

So also in the centre of gravity position, the elbow is in contact with the body, and thus the finger tips and the wrist joint are the only nodal points examined in this posture of the limbs.

The examination of the three planes at each of the chosen spots were taken in any order, but preliminary experiments showed that this did not affect the results, and consequently that the observed subject did not gain by practice at levels near each other.

Five readings at each position were originally taken by Horsley, who also made a number of observations of ten readings at each spot but found that no advantage accrued thereby, the resultant average error being practically the same. It was later found that three were really sufficient, the recording hand being completely lowered after each result had been noted; the observed finger, however, being kept in position until the three readings were finished and another plane or another level commenced.

(*a*) *The Sense of Orientation at the Points Named.*

Inasmuch as by the method detailed the person observed does not see the plate, and does not become conscious of its position until his finger has been placed upon it by the recorder or investigator, it is obvious that his knowledge of the actual relation which the plate bears to his body axes

is only obtained by his muscular and arthrodial senses "passively" stimulated. The actual tactility of the finger tip cannot be of any assistance for localisation in space, although it is of the highest value in informing the person what is in space. Hence the appreciation of orientation measured by this method is a pure mixture of muscular and arthrodial tension and pressure sensations. Tactility *per se* in the absence of sight is in fact of little topographical worth or experience to the brain without the locomotory movement which carries the tactile surface to the given point. (See "Boyle Lecture.")

We have now to refer to other strains and tension stresses set up in the limbs by the attitudes necessary to obtain the readings on the plate since these might clearly affect the results, and we will proceed to examine them in some further detail.

We have already alluded to the mechanical limitations of the method caused by the configuration of the joints of the limbs and to these we must add the actual effect of strained movements.

The first of such stresses we would mention is caused by the attitude of the limbs when the finger tip point is examined with both upper limbs fully extended. In the overhead position this attitude is definitely irksome, and possibly the considerable degree of the error of propinquity observable in this series of observations is partly attributable to the interference of "painful" impressions from the stretched muscles and tendons. Fortunately, a second and an opposing source of "stress" information tends to neutralise this complication, for our "muscular" consciousness of the position of the finger tip is improved by our knowing that it is at any given moment at the full extent of stretch of the limb, and it is probably easier then to bring the other finger into correct apposition. The rather remarkable accuracy occasionally observed at the finger tip point may be attributable to this fact, but when the general averages are taken it appears that the error of sense of position owing to the greater distance from the body governs the situation and obliterates the influence of the accessory stresses now referred to.

On adding together separately the average errors of the two index fingers obtained when the plate situated in the mesial plane of the body is held in the two chief planes at right angles to each other, namely, the sagittal and horizontal, the result shows that this muscular and arthrodial knowledge of space is unexpectedly homogeneous and of equal value for the two sides of the body. Thus:—

Right Hand Recording.		*Left Hand Recording.*	
Sagittal plane ...	86·1 cm.	*Sagittal plane* ...	90·1 cm.
Horizontal plane ...	109·8 „	*Horizontal plane* ...	105·2 „
Total ...	195·9 „	Total ...	195·3 „

On the Lines of Error in the Planes of Space in which the Observations were taken.

It has already been stated that the plate method involves the determination of the errors of the position of the recording finger at various points in each of the three planes of space, *i.e.*, vertical-sagittal, horizontal and vertical-frontal. In each of these planes the error is necessarily measured along the two principal axes of the plane which are at right angles to each other. It remains to mention the terms by which it appears to us the error of each axis may most conveniently be denoted. The ordinary anatomical expressions of anterior and posterior have relation to a vertical frontal plane passing through the longitudinal body axis, and the terms superior and inferior are in like manner employed to express higher or lower points respectively in the same frontal plane. There are also in common use the terms proximal and distal, which literally mean nearness to, or distance from, the longitudinal body axis, *i.e.*, in any plane.

(*a*) *Sagittal plane.*—Under the circumstances it follows that for observations in a sagittal plane the horizontal component of the error, if the latter be in front of the exact spot, may be described as anterior or distal, and, if behind the spot, it may be termed proximal or posterior. The vertical component of the error is always adequately

The next important limitation is fatigue; no definite investigation of fatigue has yet been made, but it was found that the consecutive taking of a large number of readings gradually tended towards an increase of error.

A very interesting limiting factor already partly alluded to is that of psychical interference. In his preliminary experiments with the plate Horsley found that whenever the person observed concentrated his attention and endeavoured to estimate by mentation the localisation of a particular point the error was frequently increased. Also that when the contrast observation was made of informing the person observed that he was wrong, and of asking him to correct it, in only relatively very few cases was the movement of correction executed in the right direction. This result we have already suggested necessarily follows from the fact that the recording finger may be moved in any direction, and the very exceptional occurrence of absolute accuracy shows that the encephalon does not gain an exact memorialisation of the space surrounding the body from the muscular and arthrodial senses.

In association with the same question of psychical interference it was noticeable through all these investigations that if the subject observed was more than usually accurate the accuracy was better maintained when his attention was not concentrated on the experiment.

(*b*) *Influence of Laterality.*

From the first, all experiments have been conducted bilaterally, *i.e.*, at each point records were taken with the two index fingers alternately observed and recording. It might have been supposed that the knowledge of the two halves of the encephalon as regards their muscular and arthrodial acquaintance with space was different, and that with right-handed people there would be a balance in favour of the left half of the encephalon. Although this is undoubtedly the case when the visual memorialisation of distance is used concurrently with the locomotion of the limbs, it is otherwise when the visual element is excluded.

Another way of stating the same fact is that the greatest accuracy is found according to the greatest ease and frequency with which the movements of the limb are habitually performed.

(*b*) *Topography of the Amount of Error according to the Aspect of the Body Examined.*

It has been known from Weber's original work that the tactility of the front and back surfaces of the body, as estimated by the compass æsthesiometer, varies very notably and that the skin of the back is as regards discrimination remarkably insensitive; also that there is a striking difference between the front and back of the forearm and hand.

On proceeding to compare the spatial orientation of the areas in front and rear of the body respectively it was very soon found that as the plate method depends on both observing and recording with the two hands the rear offered so many mechanical difficulties, *i.e.*, painful contraction of the muscles strained in reaching behind the back that a strictly parallel and complete series of readings in front and behind the body could not be carried out. But sufficient readings were obtained to indicate that the deficiencies of the pallial record of muscular and arthrodial impressions vary in much the same degree both quantitatively and regionally as those of tactility.

The combined[1] average error of each level in the sagittal and horizontal planes taken together and of both hands recording are as follows :—

Positions.		*Average error.*
(1) Front	Centre of gravity level	$\frac{5{\cdot}4}{2}$ cms.
	Enisform level	$\frac{5{\cdot}6}{2}$,,
	Shoulder level	$\frac{5{\cdot}7}{2}$,,
	Visual level	$\frac{6{\cdot}2}{2}$,,
(2) Overhead (longitudinal body axis)		... ... $\frac{7{\cdot}8}{2}$,,
(3) Behind		... $\frac{10{\cdot}4}{2}$,,

[1] *i.e.*, the errors at the same nodal points summated and averaged.

denoted as superior or inferior, *i.e.*, above or below the actual point of the plate against which the observed finger is resting.

(*b*) *Horizontal plane.*—In this plane the horizontal component of the error in the antero-posterior direction can be described as distal or proximal respectively. There remains the component of the error at right angles, namely, that on either side of the point touched, in short, the lateral error; this for observations with the plate in the mesial sagittal plane can only be described as right or left.

(*c*) *Frontal plane.*—The vertical and lateral components of the error are superior, inferior, and right or left for the two axes in this plane respectively.

It must not be forgotten that in describing the errors of experiment in points in space above the head in the longitudinal axis of the body, the terms proximal and distal have a connotation of superior and inferior, whereas their connotation is that of anterior and posterior in the other positions dealt with in this paper (see fig. 1).

I.—Results of Observations on the Mesial Sagittal Plane.

(*a*) *Concentric Increase of Error outwards from the Body.*

The first and most important generalisation which comes out clearly from comparison of the tables of the average errors (*see* Appendix A) is that there is a progressive diminution of knowledge of space as we pass outwards from the central axis of the body. This is represented proportionately by the values obtained for the various joints which show distinctly that for all levels in the sagittal plane, when the upper limbs are fully extended, the finger tips know less of position in space than when they are at the distance of the wrist, and still less than when they are at the distance of the elbow.

This would seem to suggest that our conscious appreciation of the information derived from the muscular sense and arthrodial impressions improves as the joints are more and more flexed and conversely diminishes as they are extended.

It will thus be seen that as one of the senses subserving consciousness, the muscular sense gives us fair knowledge of the space in front of the body, less of the area overhead and least behind, and that the accuracy of this spatial knowledge is best just above the centre of gravity of the whole body from which point it diminishes as we pass up the body towards the head and over on to the back, thus corresponding in general with the distribution of tactility on the surface of the trunk.

(*c*) *Topography of Amount of Error according to the Neighbourhood of the Centre of Gravity of the whole Body.*

The above list of the values of the total error in any given position reveals a much more interesting point than the simple correlation between the musculo-arthrodial sense and tactility we have just described, and that is the unexpected fact that the degree of error steadily diminishes from above down until it reaches its minimum about the level of the centre of gravity of the whole body. This can only mean that our muscular and arthrodial sense impressions have in their pallial representation a focus, or central point, in space around which and in co-ordination with which they are grouped.

That the development and functional efficiency of the muscular sense should bear a relation to the centre of gravity of the whole body seems quite reasonable when we consider that the centre of gravity is the true focus of the cerebral representation of equilibrium, and that of necessity the memories of movements must include not only the co-ordinated sequences of muscular contractions, but also the degree and relation of these latter to the centre of gravity of the mass of the body for the conservation at the moment of the latter's position in space.

(*d*) *Topographical Determination of the Direction of the Error.*

(1) *Propinquity and distality.*—An additional and prominent reason for originally introducing the plate test was

to secure more accurate and numerical determinations of normal topognosis, since that sense is particularly liable to be disturbed and indeed abrogated by lesions of Rolandic zone.[1]

Horsley showed[2] that the characteristic sensory error in cases of loss of function of the pallial Rolandic zone is propinquity, *i.e.*, the person tested topognostically indicates a spot nearer the body than the point actually touched when the part explored is the dorsum of the hand and forearm and the stimulus is a light touch[2] on the skin.

To judge correctly of this pathological condition it is necessary to know whether in normal individuals there is any error of propinquity in the exercise of the muscular sense, associated as that is in the plate test also with pressure tactility.

Obviously this point only appears in the examination of the sagittal and horizontal planes, so far as the front and back of the body are concerned, while in the overhead measurements an error of propinquity or nearness only comes in with the frontal and sagittal spatial planes.

The error of the frontal plane not being under consideration in the present paper, we have taken the whole series of records of the error when the plate is in the horizontal and sagittal planes. The number of times in which the error was in the direction of propinquity, or nearness to the body, was 645, and the number of times it was distal was 407, a relation between the two of 12 to 7·5 in favour of propinquity. It is thus clear that a topognostic error of propinquity is a normal condition, and is greatly exaggerated under the circumstances of disease.

The records also show that the error of propinquity is much more frequently committed when the plate is in a sagittal plane than horizontal—in fact, the error of propinquity in the sagittal test is almost twice that of the horizontal, the actual figures being 15 : 8. The greatest

[1] This expression, for the sake of argument and in the absence of more precise information under the heading of function, must be taken to mean both central, *i.e.*, frontal and parietal gyri.

[2] *i.e.*, with strict exclusion of pressure stimuli in a moderately marked case. In a severe case the error occurs in the case of pressure and even pin prick stimuli.—V. H.

error was found to be committed when the plate was examined sagitally in overhead positions—in fact, the proximal character of the overhead error is so great as to raise materially the average when all results are taken together.

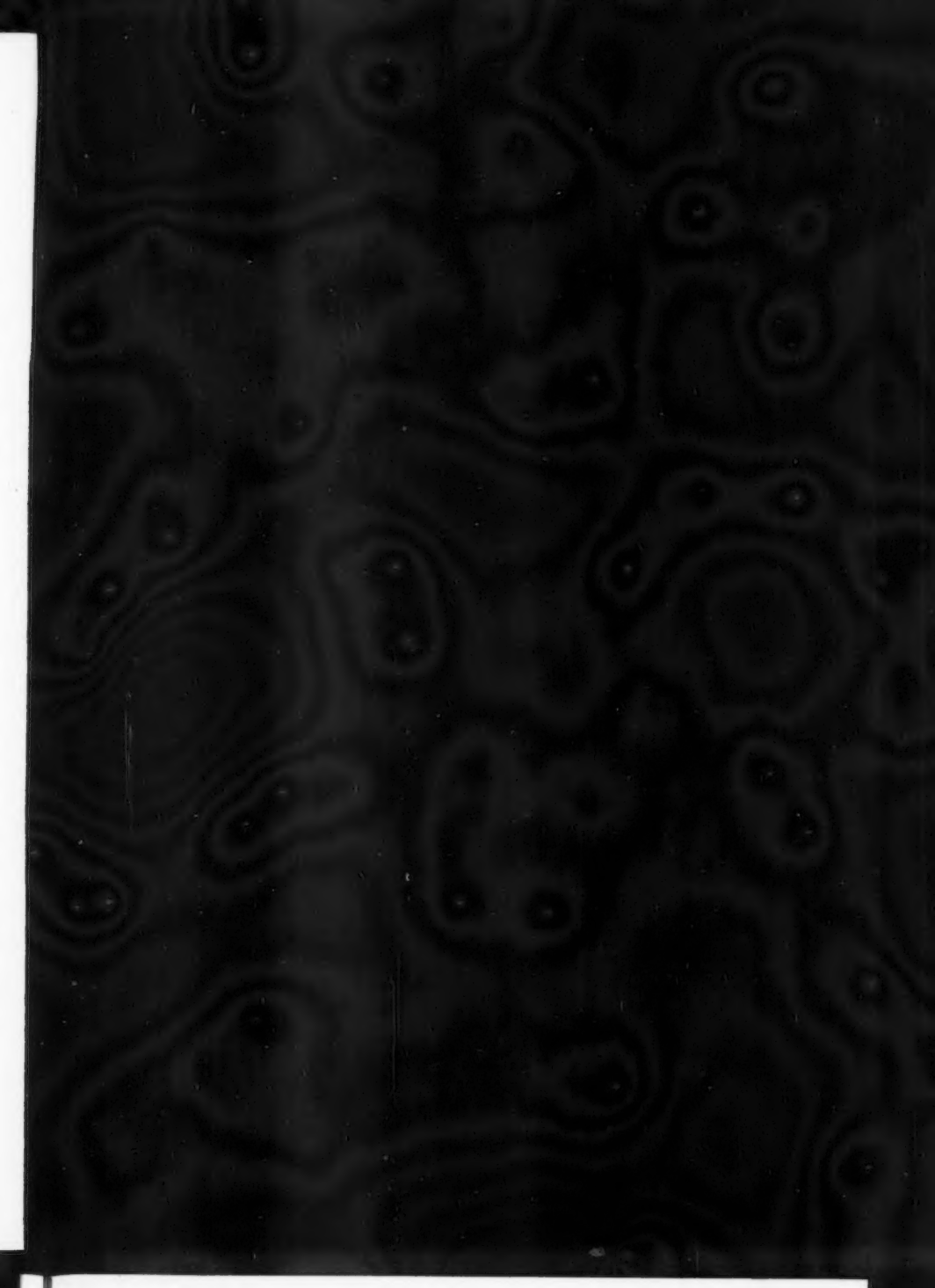
If the "overhead" results are excerpted, then the relative greater frequency of propinquity, or proximal error, is so diminished as to give its almost invariable occurrence in cases of cerebral disease a higher interest and importance.

It may be useful for clinical work to give a few absolute determinations of the normal amount of the error of proximality or propinquity. For this purpose we take as probably the readiest position for clinical investigation the "wrist" point in the "ensiform" level, and we add together the results of the horizontal and sagittal planes. The averages observed are for proximality $\frac{2{\cdot}6}{2}$ cm., or, roughly, 1·5 cm., and for distality $\frac{2{\cdot}2}{2}$ cm., or, roughly, 1·25 cm. These figures, therefore, may be used as a basis for examining cases of pathological disturbance of the muscular and arthrodial senses, but it is necessary to point out that the standard now offered is founded on averages, and that similarly any series of pathological cases for absolute comparison must include a sufficient number of observations to provide a true average before conclusions can be justly drawn as to the character of the error found.

(2) *Superiority and inferiority.*—Having discussed the horizontal component of the direction of the error under (1), we are now in a position to observe what error is made in the vertical plane from above down, or, to use a common anatomical expression, superior or inferior.

Speaking generally, we may say at once that overhead there is a very strong tendency to an error of "inferiority;" this, of course, in this position is the same as propinquity, and has already been mentioned; we turn, therefore, to the observations made at the various positions in front of the body where the following striking result was observed.

The vertical component of inferiority steadily diminishes from above down as the points are successively recorded, until the interval between the ensiform and centre of



The following is an example of the Tables which hav

MESIAL SAGITTAL

Initials and Number of Times Tested.	I.			II.	
	a	*b*	*c*	*a*	*b*
V. H. 1	14·1 A I	18·6 A I	7·6 A I	9·5 P I	9·4 P I
H. 1	11·2 A I	10·4 P I	5 0 P S	5·6 P S	9·3 P S
C. 1	16·6 A I	15·2 A S	10·6 A S	7·8 P I	4·8 D I
F. 1	6·1 A I	10·2 A I	5·7 A I	4·3 O I	8·8 P I
S. 1	13·9 A I	8·2 A I	8·5 A S	10·7 D I	4·4 D I
C. 2	9·5 A I	4·1 P I	3·0 A S	5·6 D I	5·0 D I
H. 2	5·0 P S	7·6 A S	4·5 A I	1·8 P S	6·0 D I
G. H. 1	19·8 A I	24·2 A I	8·9 A I	15·4 D I	11·0 D I
R. 1	12·5 A I	11·8 A I	8·0 A I	5·3 P I	2·3 D S
R. 2	7·0 P I	6·0 P I	4·9 P I	8·8 P S	1·5 D I
M. 1	———	———	———	2·6 P I	9·8 D I
R. 3	17·6 P I	9·8 P I	5·9 A I	7·3 P S	4·4 P S
B. 1	13·9 A I	6·0 A I	3·4 P I	2·7 P S	5·2 P I
B. 2	4·0 P I	6·0 P I	7·0 A I	2·4 P I	2·5 P S
S. 2	20·8 P I	15·3 A I	3·6 P S	3·7 D I	2·3 P S
F. 2	8·0 A I	7·0 A I	3·0 P S	7·1 P I	7·4 D I
M. 2	2·2 A I	6·0 A I	7·0 P I	3·2 P S	2·5 P S
S. 3	6·4 A I	14·0 A I	4·4 A I	3·8 D I	3·0 D I
C. 3	6·0 P S	1·6 P I	3·3 A S	1·4 D O	7·4 P S
L. 1	4·0 A I	8·0 P I	3·4 A S	2·2 P I	3·0 P S
I. 1	2·4 P I	5·1 A I	2·3 A I	5·1 P I	2·5 P I
R. 4	8·1 P I	10·0 P I	9·0 P I	1·1 O S	3·0 D I
S. 4	8·0 A I	13·0 A I	10·7 A I	5·2 D I	2·8 P I
R. 5	7·0 P I	5·8 P I	5·7 P I	2·8 P I	3·0 D I
S. 5	15·8 A I	12·1 A I	9·5 A I	7·0 D I	4·1 P I
	239·9	236·0	144 9	132·4	125·4
	10	10	6	5·3	5

A = Anterior. I = I

Appendix A.

e Tables which have been constructed from the results. The Table here given sufficiently shows the general tendency of variation.

ESIAL SAGITTAL PLANE. ERROR IN $\frac{\text{CMS.}}{2}$. PLATE SAGITTAL. L. HAND RECORDING.

II.		III.			IV.			V.		VI.
	b	*c*	*a*	*b*	*c*	*a*	*b*	*c*	*a*	*b*	
I ...	9·4 P I	... 6·6 P S	... 5·0 P S	... 6·6 P I	... 2·4 D I	... 9·6 P S	... 8·2 P S	... 4·5 D S	... 3·8 D S	... 4·5 D S	... 17·6 P I
S ...	9·3 P S	... 5 2 P S	... 3·9 D S	... 7·9 P S	... 4·0 D S	... 5·0 P S	... 6·8 D S	... 6·5 D S	... 6·4 P S	... 7·2 D S	... ———
I ...	4·8 D I	... 14·0 D S	... 13·0 P I	... 8·5 D I	... 10·7 D I	... 11·0 P I	... 5·3 D I	... 8·1 D I	... 9·0 P I	... 2·1 P I	... 10·0 P I
I ...	8·8 P I	... 3·5 P I	... 6·9 P I	... 6·2 P I	... 5·0 P O	... 8·1 P S	... 3·3 P S	... 1·5 D S	... 5·4 P I	... 10·0 D S	... 6·0 P I
I ...	4·4 D I	... 5·0 D I	... 4·7 P S	... 7·2 D I	... 4·4 D I	... 6·7 D I	... 6 0 D I	... 4·7 D I	... 4·4 D I	... 8·5 D I	... 15·6 D I
I ...	5·0 D I	... 4·2 P S	... 8·5 O I	... 5·8 D I	... 3·1 D I	... 4·9 D I	... 3·2 D I	... 5 0 D I	... 5·5 P I	... 4·2 D I	... 4·0 P S
S ...	6·0 D I	... 4·2 D S	... 2·0 P S	... 6·9 D I	... 6·3 D I	... 5·2 P I	... 4·2 P S	... 5·0 D I	... 2·8 O I	... 4·8 D I	... 10·0 D I
I ...	11·0 D I	... 9·4 D I	... 10·1 P I	... 5·1 O I	... 5·6 D I	... 6·8 P I	... 3·6 P I	... 6·0 D I	... 3·7 P I	... 1·9 P I	... 4·2 D I
I ...	2·3 D S	... 6·4 D I	... 3·3 P I	... 3·0 D I	... 8·0 D I	.. 10·3 P I	... 9·3 P I	... 1·8 D I	... 9·1 P I	... 6·2 P I	... 10·0 P I
S ...	1·5 D I	... 2·8 D I	... 6·7 P S	... 5·4 P S	... 1·5 P I	... 3·5 P S	... 5·3 P S	... 4·2 D S	... 4·3 P S	... 3·3 D S	.. 23·0 P I
I ...	9·8 D I	.. 6·4 D I	... 9·6 D I	... 6·0 D I	... 2·2 P S	... 2·4 D S	... 3·0 D S	.. 3·0 D S	... 5·3 D O	... 5·8 D S	... 9·3 D I
S ...	4·4 P S	... 3·4 P S	... 3·6 P S	... 4·7 P S	... 5·4 D I	... 2·3 D S	... 4·1 P S	... 6·2 D S	... 2·4 P S	... 6·0 D S	.. 19·8 P I
S ...	5·2 P I	... 6·8 P I	... 6·0 P I	... 5·8 P I	... 1·1 O I	... 4·7 P I	... 2·5 P I	... 2·0 D S	... 3·2 P I	... 2·5 P S	... 9·4 P I
I ...	2·5 P S	... 4·1 P I	... 8·0 P S	... 1·4 D I	... 4·0 D S	... 9·0 P S	... 4·4 P I	... 1·9 P I	... 4·6 P S	... 1·6 P S	... 22·1 P I
I ...	2·3 P S	... 2·8 D I	... 9·2 D I	... 4·0 P S	... 2·5 P S	.. 4·0 P I	... 4·9 P I	... 4·4 D I	.. 4·2 D I	... 1·0 D I	... 13·1 D I
I ...	7·4 D I	... 8·0 D I	... 1·9 P I	... 8·5 P I	... 1·0 P I	... 3·1 P S	... 3·4 P S	... 2·5 P I	... 3·1 D O	... 1·1 D I	... 9·5 D S
S ...	2·5 P S	... 2·3 P S	... 2·6 P S	... 3·1 O S	... 1·4 P I	... 2·2 P I	... 1·3 O S	... 2·2 P I	... 3·6 O S	... 2·8 D S	... 6·2 P I
I ...	3·0 D 1	... 5·0 D I	... 1·4 D O	.. 6·8 P I	... 3·8 D I	... 7·4 P I	... 8·4 D I	... 5·5 D I	... 5·6 D I	... 3·5 P I	.. 8·4 D I
O ...	7·4 P S	... 4·1 P S	... 6·1 D I	.. 2·4 O S	... 3·1 P S	... 2·7 D S	... 6·3 P S	... 2·4 P S	... 4·4 D S	... 4·0 D S	... 3·1 P S
I ...	3·0 P S	... 0·9 D S	.. 2·4 P I	... 1·8 P S	... 1·1 D S	... 6·1 P S	... 2·2 D S	... 1·6 D S	... 4·7 D S	... 3·8 D S	... 14·4 P I
I ...	2·5 P I	... 3·4 P I	... 2·1 P I	... 2·0 O I	... 1·8 P I	... 3·0 P I	... 0·4 D S	... 1·3 P I	... 3·6 P S	... 3·8 D S	... 15·0 P I
S ...	3·0 D I	... 3·4 D S	... 2·7 D I	... 2·4 D S	... 2·4 D S	... 3·5 P S	... 2·5 D I	... 3·5 P S	... 3·6 P S	... 1·9 P S	... 7·8 D I
I ...	2·8 P I	... 5·2 D I	... 8·1 D I	... 7·1 P I	... 4·5 D I	... 11·0 D I	... 8·1 P 1	... 5·5 P I	... 3·2 P I	... 6·2 P I	... 6·7 P S
I ...	3·0 D I	... 1·7 D I	... 4·4 P I	... 3·7 P I	... 3·1 D I	... 5·6 P I	... 8·1 P I	... 2·4 P I	... 5·4 D S	... 2·5 D S	... 9·5 P I
I ...	4·1 P I	... 7·6 D I	... 3·1 D I	... 9·6 P I	... 5·2 D I	... 7·1 P I	... 7·5 D I	... 6·3 D I	... 2·3 P I	... 3·4 D I	... 5·1 D I
...	125·4	... 126·4	... 135·3	... 131·9	... 93·6	... 145·2	... 122·3	... 98·0	... 123·1	... 102·6	... 259·8
...	5	... 5	... 5·4	... 5·3	... 3·7	... 5·8	... 4·9	... 3·9	... 4·9	... 4·1	... 10·8

= Anterior. I = Inferior. S = Superior. P = Proximal. D = Distal. O = No difference.

gravity levels is reached; here the two tendencies of marking high and marking low neutralise each other.

The greater tendency to the error of "inferiority" is shown by summing together the total number of observations as follows:—

Frequency (percentage) of occurrence of superiority and inferiority.

Left Hand Recording.	*Right Hand Recording.*
Superior = 47 per cent.	Superior = 48 per cent.
Inferior = 53 per cent.	Inferior = 52 per cent.

The same facts also show that, although there is often a strongly-marked difference between the two hands (*i.e.*, the right often makes a superior error compared to the left), nevertheless, this difference disappears when all the results are averaged together.

We will conclude this paragraph by stating that on summating the averages, the absolute amount of the error of inferiority for the levels from above down varied from 2·5 cm. to 1·5 cm.

(3) *Right and left.*—We must now discuss the laterality of error observed when the plate is horizontal.

As before stated, when the recording index finger is placed to one side or other of the observed finger, since the latter is in the mesial (vertical) plane of the body, such lateral (horizontal) deviation of the recording finger can be properly denoted by the terms Right and Left.

Taking first the number of times in which such lateral deviation was observed, a very noteworthy feature of tendency to cross the two hands was found to exist. Thus, of the total number of cases, the result was as follows:—

Crossing Middle Line.

Right hand recording	...	Left = 61 per cent. Right = 39 per cent.
Left hand recording	...	Right = 68 per cent. Left = 32 per cent.

It will thus be seen that both hands tend to pass beyond the actual point aimed at, and of the two hands this was

slightly more pronounced in the left. It may here be observed that of the subject of experiment not one was strictly left-handed.

As regards the frequency with which this error of laterality was committed, the figures show that it followed the same law as that of superiority and inferiority—namely, that it was most marked at the shoulder level and least marked at the centre of gravity. In fact, at the centre of gravity level there was a slight majority of cases in which, with either left or right hand recording crossing did not occur at all.

This result was also confirmed by the average amounts of the error at the different levels.

To give now absolute measurements, these we found to vary on the average from 3·5 to 2 cm. for the left hand crossing to the right and 4 cm. to 2 cm. for the right hand crossing to left. From this it would appear that, though the right hand crosses fewer times than the left, when it does so the error is quantitatively greater.

There are several minor, but not uninteresting, points to be considered, especially in regard to this question, of one hand crossing the mesial plane of the body when the plate is fixed horizontally.

It may be as well to first remark here that in general the results obtained with the plate horizontal as compared with those with the plate sagittal show a slight increase of error at all levels. This we would ascribe to the fact that the two limbs are in exactly the same position when the plate is placed sagittally ; whereas when the plate is horizontal, the observed hand being placed on the under surface of the glass is fully supinated, whilst the recording hand being moved down to the upper surface of plate must be fully pronated. Hence in the sagittal cases the sensations of homologous muscles in the two arms are involved, whilst in the latter those from two different groups of muscles are concerned. It may be added in furtherance of this view that in testing with the plate frontal where the error is still greater the postures of the limbs are also more dissimilar.

An interesting outcome of the observations, showing that the majority of cases exhibit a crossing of the two limbs, is the fact that most persons when tested remark on the feeling of great thickness of the plate when it is placed sagittally, and are subsequently surprised to find it much thinner than they expected. The recording hand being prevented from crossing over the middle line by the plate placed sagittally in the mesial body plane seems to suggest to them that the plate keeps the hands a long way apart and hence feels very thick; this feeling is of course most marked in those cases in whom the crossing occurs most often and to a marked degree. Another interesting point is that a few individuals show a tendency to make a larger lateral ("crossing") error with one recording hand than with the other, the plate being horizontal. If now the plate is placed sagittally in the mesial sagittal plane such persons imagine it to be placed nearer to that side on which their lateral error was greatest. This shows that there is functional asymmetry of the pallial representation of orientation analogous to Guldberg's observations of structural asymmetry as affecting orientation.

(*e*) *The Error when the Observations are made behind the Body.*

Owing to the causes mentioned above, the readings behind the body show the largest error, and even here, on the averages, a fall of error from above down was seen in a few cases examined with the plate in different levels behind the body. The strained positions, however, so complicated the direction of error that nothing more specific could be deduced from these observations.

II.—Results of Observations on Lateral Planes (Nipple and Shoulder).

(*a*) *General.*—After the mesial sagittal plane had been examined with the results shown above, normal individuals were examined in the vertical lateral plane passing through the shoulder joint in the same manner, when it was found

that in this, as in the mesial plane, there was the same fall in the amount of error from the (finger tip) periphery towards the body and also in the successive levels from above down towards the centre of gravity. Since a complete examination of many lateral planes in all positions of the plate would have necessitated an excessive number of observations on each subject, on several successive days, other errors, such as fatigue, would have been introduced and from the monotony probably lack of care in recording on the part of the subject. It therefore seemed better to take for this purpose planes in one level only, namely, the "ensiform," especially as this appeared likely to prove the most convenient level for clinical work, and, after investigating selected lateral planes in each subject at this level, to compare the results with those obtained at the same level in the mesial sagittal plane on the same day.

The lateral planes chosen were one through each nipple, and one through the centre of each shoulder joint, these planes being sagittal and parallel therefore to the mesial sagittal one.

The three points in space or distances from the body surface mentioned before were used in each of these planes, namely, the finger tip, wrist and elbow, and thus comparable results were obtained.

On adding together the averages of the three positions of both hands recording, and with both the plate sagittal and horizontal in each plane at the "ensiform" level, the following numbers in half cms. were obtained:—

	Right shoulder plane.	Right nipple plane.	Mesial sagittal plane.	Left nipple plane.	Left shoulder plane.
Half cms.	73·7	67·2	56·9	67·9	73·8

It will thus be seen that the error increases the further the plane examined is from the mesial sagittal plane, and that this error is the same on both sides when comparing similar planes. This is interesting because it shows that there is no substantial increase of accuracy in the planes on the right side as might *a priori* have been expected. Each plane also showed well the fall of error as the body was

approached. It will be remembered that the error in the mesial sagittal plane with the right hand recording was equal to that made by the left hand recording on adding together the results obtained at all the levels—this is again seen in the ensiform level of the mesial sagittal plane in this series, where the numbers $\frac{28{\cdot}3}{2}$ were obtained for the right hand and $\frac{28{\cdot}6}{2}$ for the left.

Ensiform Levels.

	Right shoulder plane.	Right nipple plane.	Mesial sagittal plane.	Left nipple plane.	Left shoulder plane.
Right recording ...	38·0	34·1	28·3	31·9	35·3
	Left shoulder plane.	**Left nipple plane.**	**Mesial sagittal plane.**	**Right nipple plane.**	**Right shoulder plane.**
Left recording ...	38·5	36·1	28·6	33·1	35·7

In comparing the errors made with the right hand recording in, say, the left nipple plane, this must be compared with the left recording in the right nipple plane and *vice versâ*, it will thus be seen that the right hand is slightly more accurate in the right nipple plane than the left hand is in the left nipple plane, and the right hand is again slightly more accurate in the left nipple plane than the left hand is in the right nipple plane. In both places the error is about 1 cm. in favour of the right hand, but it must be remembered that this is taken from six positions, hence this only gives the right hand a superiority of ·16 cm. in 2·6 cms. over the left at each position—and, finally, when it is remembered that these readings are the averages from fifty cases, the difference is clearly very small.

It will be noticed that the number obtained by the right hand recording in the right lateral planes and the left recording in the left lateral planes are larger than those obtained when the recording hand crosses over to planes on the opposite side of the body. The fall of error from the periphery towards the body showed some variations, also when the hand was recording in planes on its own side of the body, and these variations became more marked the further the planes were from the middle line—these we

ascribe to the awkwardness and contracted limitation of the movements of the recording hand. The elbow position in particular in these most lateral planes showed a larger error than could be otherwise explained, and the attitude of the limb in this position was also felt to be irksome by the subjects investigated.

(*b*) *Propinquity and distality.*—In addition to the general predominance of the error of proximality or propinquity, some interesting results were obtained in the relative number of times the errors of propinquity or distality were made and according to the lateral plane examined. When a "shoulder" plane was examined with the hand of that side recording, a large number of cases showed a large percentage of errors of distality, this was less marked in the nipple plane of that side, and the error became more and more proximal until the opposite shoulder plane was reached.

Right Hand Recording—Percentage of Proximality.

Plane.	Right shoulder plane.	Right nipple plane.	Mesial sagittal plane.	Left nipple plane.	Left shoulder plane.
Vertical sagittal	31 %	36 %	49 %	64 %	68 %
Horizontal	28 %	36 %	44 %	67 %	63 %

Left Hand Recording—Percentage of Proximality.

	Right shoulder plane.	Right nipple plane.	Mesial sagittal plane.	Left nipple plane.	Left shoulder plane.
Horizontal	67 %	62 %	59 %	45 %	39 %
Vertical sagittal	66 %	72 %	60 %	46 %	30 %

This fact comes out even more clearly when the results are stated graphically (see fig. 3).

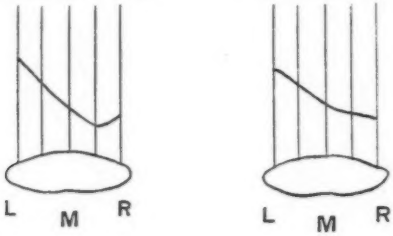
To give now some absolute numbers, the average error of propinquity taken from all the cases with both hands recording and ensiform wrist position in the sagittal and horizontal plate in the mesial sagittal plane was $\frac{2 \cdot 6 6}{2}$ cm. or 1·5 cm. roughly—that of distality was $\frac{2 \cdot 3 7}{2}$ or 1·25 cm.; in the lateral planes this average was increased by ·6 cm. more.

(*c*) *Superiority and inferiority.*—As the levels taken in the lateral planes were in the same horizontal line, the relative number of times the error was superior as against the number of times it was inferior, show no marked

Graphic representation of the relative percentage of Propinquity and Distality in Lateral Planes.

LEFT HAND RECORDING.

Plate vertical and sagittal. Plate horizontal.



Relative Percentage of Propinquity and Distality in Lateral Planes.

RIGHT HAND RECORDING.

Plate vertical and sagittal. Plate horizontal.

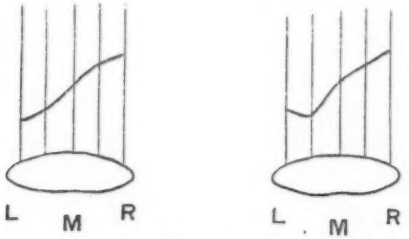


FIG. 3.

The ellipsoidal figure represents the outline of the transverse section of the trunk, and the straight lines the imaginary sections of the sagittal planes examined, namely, shoulder, nipple and mid-line. The curved lines show the gradual fall of the percentage, and are obtained by taking the length of the straight line as 100 and measuring thereon the value of the error in each case.

M = Mesial sagittal plane. L = Left. R = Right.

variation. As was observed when examining the levels from above down, they remain approximately the same, only showing small irregular fluctuations for which no cause could be found.

We will therefore give some of the average errors of superiority and inferiority at the "ensiform-wrist" positions in the mesial sagittal planes for comparison with those in the lateral. Thus, while the average "superior" error in the mesial sagittal plane was $\frac{3\cdot3}{2}$, or 1·6 cm., the inferior error being $\frac{3\cdot6}{2}$, or 1·8 cm., the lateral planes showed an increase of error amounting to ·8 cm. more.

(*d*) *Right and left.*—It has already been shown that the tendency for the hands to cross in the mesial sagittal plane is very great, and it might have been anticipated that this would be lessened in the lateral planes on account of the increasing limitation of movement of the recording limb. This seemed still more probable as it was observed to occur in a few pathological cases in whom Horsley had removed tumours from the Rolandic zone. Thus, in these instances the crossing became less and less the more laterally the plate was placed until no crossing occurred at the shoulder plane. In normal persons, however, this is not so, the crossing becoming more marked the more laterally the plate is moved.

In examining such crossing of the record of the right hand in the left planes it must be compared, of course, with that of the left hand in the right planes and conversely.

The number of times crossing occurred is as follows :—

Planes.

	Left shoulder.	Left nipple.	Mesial sagittal.	Right nipple.	Right shoulder.
Left hand recording	78 %	66 %	64 %	61 %	66 %
	Right shoulder.	Right nipple.	Mesial sagittal.	Left nipple.	Left shoulder.
Right hand recording	72 %	75 %	67 %	74 %	78 %

Taking all the equivalent planes together to eliminate accidental variations we get crossing at each in relation to the mid-axis of the body in the following percentages of the observations.

Mesial sagittal plane.	Nipple planes.	Shoulder planes.
65·5 %	69 %	73·5 %

It is important to note that some cases showed hardly any tendency to cross, and in these persons error in any direction was always smaller than the average.

To compare average absolute numbers in the "ensiform-wrist" position, the average readings in the mesial sagittal plane were 2·4 cm. for the left hand crossing to the right, and 2·7 cm. for the right hand crossing to the left, while in the lateral planes these figures are increased by another centimetre.

III.—Results of Observations on Blind Persons.

After the figures for the mesial sagittal plane had been obtained in normal individuals it was thought that application of the plate test in cases of long-standing blindness might give additional means of control, and lead to interesting results. For this purpose twenty-two adolescents and young adults who had become blind soon (6 months to 4 years) after birth were observed. This opportunity we gained through the great kindness of Dr. Campbell, Principal of the Normal College for the Blind, South Norwood. Every opportunity was afforded by him to Dr. Slinger to make the requisite observations, and the blind students evinced much interest in the work.

In a first series eight cases were examined with the plate vertical, in the mesial sagittal plane, the ensiform level being chosen, and in each case records were taken at the finger tip, wrist and elbow positions, the hands recording alternately as heretofore.

The following results were obtained. On comparing the total averages for this level (ensiform) with those obtained previously from normal cases, it was found the blind cases showed a diminution of the normal error by about one-fourth, the actual ratio being as ·74 : 1, the normal cases being taken as unity, the added average errors being:—

Blind.	Normal.
$\frac{20\cdot7}{2}$ cm.	$\frac{27\cdot8}{2}$ cm.

The above ratio was found to be constant when the averages at all the different positions were compared, so that the greater accuracy was not merely a characteristic of any

one position, but was a feature due to the greater muscular and arthrodial sense accuracy of the blind.

As regards the laws governing the incidence of error generally, the blind showed the same concentric fall of error as the plate was moved from the finger tip position towards the body as in normal cases.

Further, the direction of error was the same as in the normal individuals, both as regards proximality and distality, and superiority and inferiority.

A second series of readings were taken from thirteen cases, the plate being fixed at the ensiform level as before, but placed horizontally.

In this series, again, the diminution of error was in favour of the blind, the ratio blind to normal being ·72 : 1, thus closely agreeing with the result of the other series.

The actual added averages were :—

Blind.	Normal.
$\frac{28{\cdot}44}{2}$ cm.	$\frac{39{\cdot}4}{2}$ cm.

It will be seen from these figures, in which the results were obtained with the plate horizontal, that they show a greater error than with the plate sagittal. Thus, in this fundamental principle also the results in the blind fully correspond to those found in normal individuals.

Moreover, the error with the horizontal plate followed the same direction as in normal cases, the crossing being marked as before. The blind (at least in the cases observed) showed a tendency to cross more often with the right hand recording than with the left. The relative frequency of propinquity and distality corresponded quite closely with that seen in normal persons.

The general deductions from these observations are of considerable interest, for as a control of the method they show that the plate is a thoroughly adequate means of estimating orientation as far as it is based on the muscular and arthrodial senses ; they also exhibit in a very clear manner how in a normal person the visual and muscular senses habitually supplement each other, so that, if the information gained by sight is permanently blotted out, the muscular sense under necessity can, by education, be

brought to a point at least one-fourth better than that learnt by a normal seeing individual. In confirmation of this conclusion the plate test showed in a very striking manner that the pupils who were completing the technical training of the College were notably superior in this orientative exercise of their muscle sense compared to those who were just entering on their special education.

The remarkable results attained by Principal Campbell are thus due to the assiduous care with which he has directed the training of the students on the lines of perfecting their memorialisation of movements.

Striking as are the results he has obtained, it is of perhaps especial neurological interest to find that the physiological groundwork of the encephalic (*i.e.*, cerebral and cerebellar) representation of such memories is unaffected by the skill of the teacher.

IV.—Conclusions.

(1) The faculty of orientation in space, as determined by the muscular and arthrodial sense, progressively diminishes from the surface of the body outwards to the limits of the arm extended in any direction.

(2) Orientation knowledge increases in passing from point to point in the space around the body, beginning above the head, coming down to the front of the body, and gradually approaching the centre of gravity of the whole body.

(3) Orientation knowledge also increases in passing from point to point in the space around the body, beginning laterally, *e.g.*, in the plane of the shoulder, and approaching the mesial sagittal plane of the body.

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NOTE ON THE TÆNIA PONTIS.

BY SIR VICTOR HORSLEY, F.R.S., F.R.C.S.

(*From the Laboratory of Clinical Pathology, University College, London*).

(1) General Anatomical Introduction.

Running along the frontal border of the pons varolii is a bundle of fibres which, when developed on the external surface of the mesencephalon, appears to emerge from the pons where the mesial border of the crus cerebri is lost in the interpeduncular ganglion. The tract runs dorsally and caudally over the crus and lower third of the lateral fillet to reach the outer border of the superior cerebellar peduncle. Guided by the dorsal surface of the peduncle, the bundle reaches the anterior border of the cerebellar cortex just where the primary fissure (Elliott Smith) debouches on the free surface of the superior and middle peduncle. This tract, to which Henle gave the name of tænia pontis, has customarily been regarded as an aberrant part of the pons, though the origin, destination and true direction of its fibres are as yet unknown.

Some facts bearing on these points have gradually accumulated in my records, and may now perhaps be worth publication.

(2) Comparative Morphology.

The individual and comparative anatomy of the tænia pontis are both interesting. In the first place, in all genera of animals in which it is obvious it is rarely symmetrically exposed on the surface of the mesencephalon, and in the very large majority it is in fact imbedded in the lateral wall of the mesencephalon and lateral fillet region (*vide infra*).

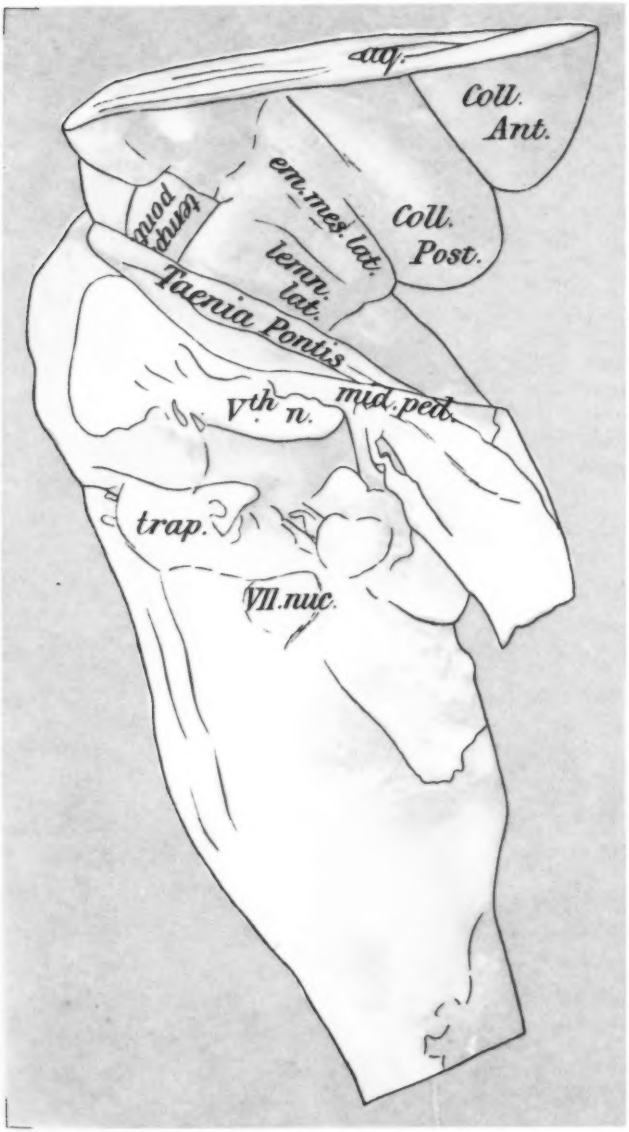
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FIG. 1.

Brain Stem of the Camel. $\frac{1 \cdot 2 5}{1}$.

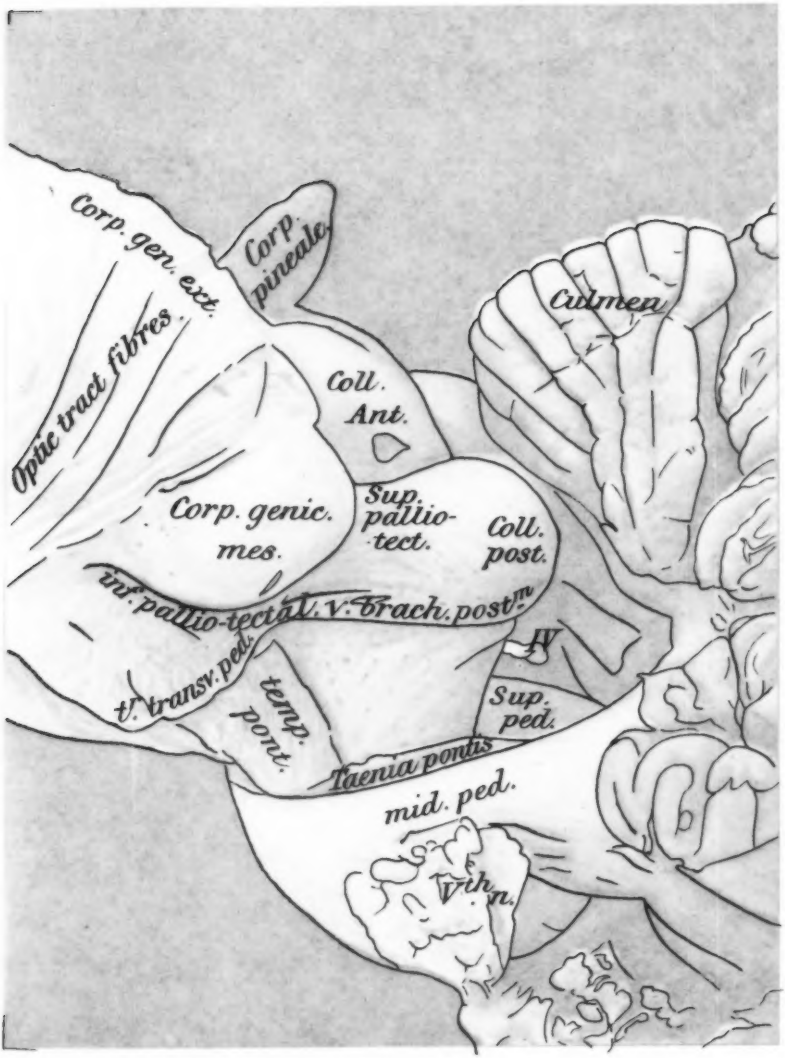


Fig. 2

Brain Stem of the Hippopotamus. $\frac{2}{3}$

Corp. pen. ext.
Corp pineale
Opt. tract. fibres
Coll. Ant.
Corp. penic. mes.
Sup pallio- tect.
Coll. post.
Inf. pallio-tect. v. Strach. post.
Inf. brach. ped.
Sup. ped.
Corp. pont.
Tegm. pontis
Mid. ped.
Pyr.
Cutaneo

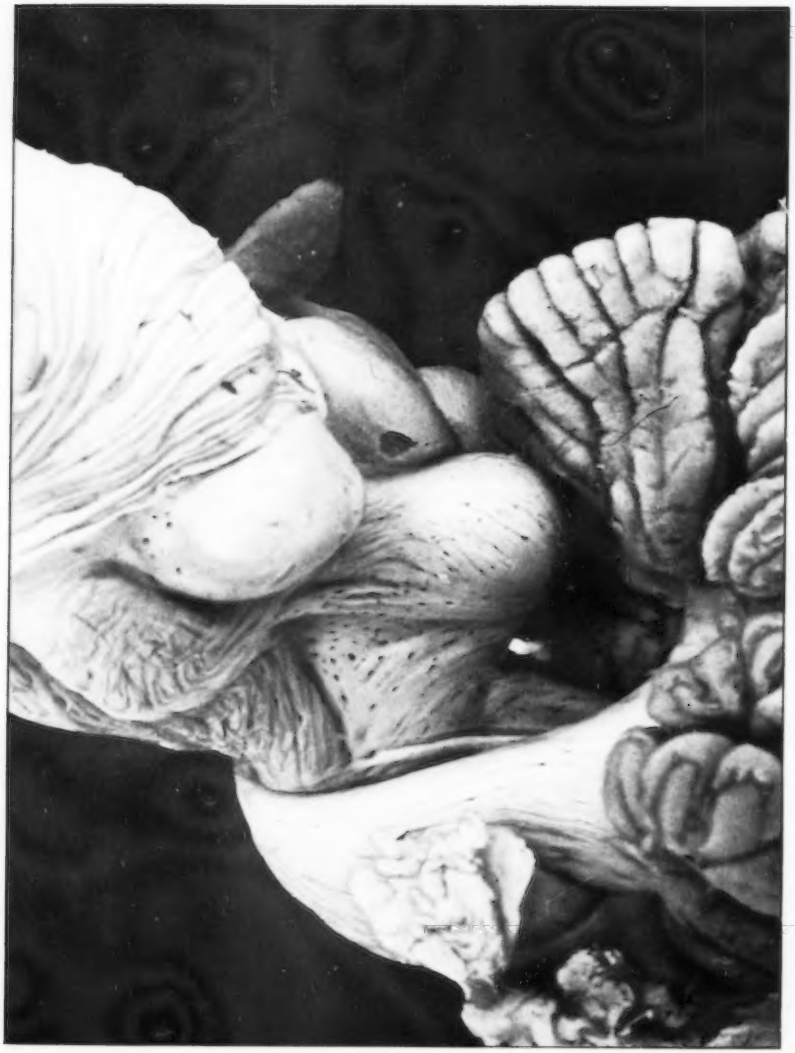
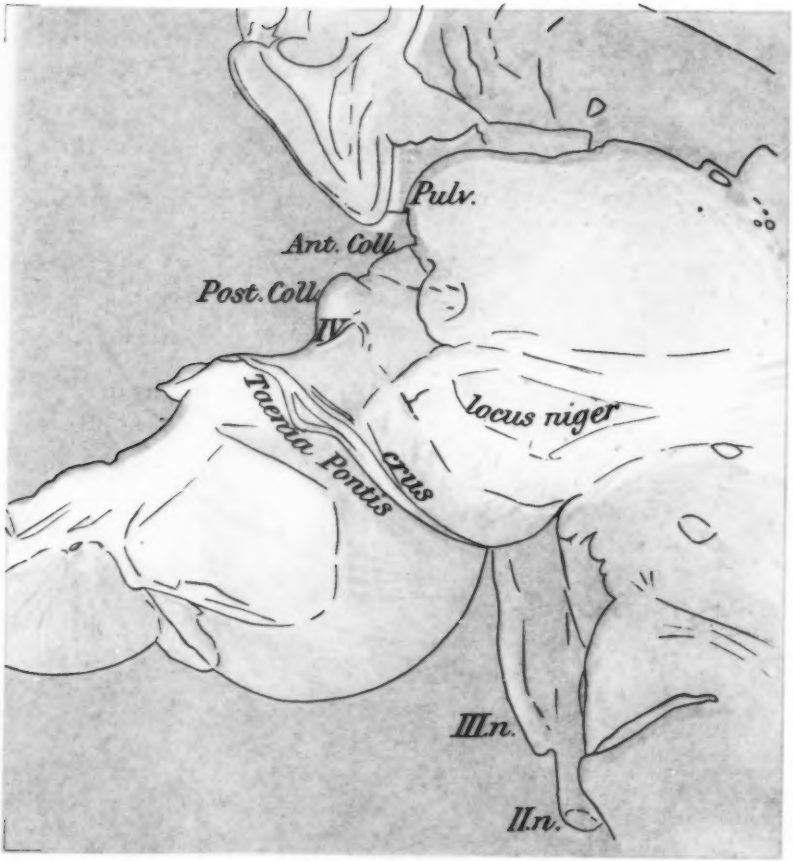


FIG. 2.

Brain Stem of the Hippopotamus. $\frac{2}{1}$.


Pulv.
Ant. Coll.
Post. Coll.
IV
Taenia Pontis
crus
locus niger
III.n.
II.n.

Pulv.
Ant. Coll.
Post. Coll.
IV
Tectum
Pons
Crus
Locus niger
III n.
II n.

FIG. 3.

Brain Stem of Man. (Case of Microgyria.) $\frac{2}{1}$.

In the second place, it more frequently tends to lie superficially as the zoological scale is successively descended, but as far as I have seen, it is more exposed in the ungulata than in other classes.

The photographs (figs. 1, 2, 3) show the tract in the camel, hippopotamus, and man.

It will be seen that in all animals the course of the tract is the same, and that the only structural variation is the relatively unimportant fact that the tract is often sub-divided into two or more bundles, instead of constituting one larger fasciculus.

At the same time the fact of its asymmetrical development, even in the quadrupedal ungulates deserves some attention, since it is reasonable to surmise that since, as we shall presently see, the tract is a cerebello-petal one, it may have an analogous function to the middle peduncle, and be concerned in the orientation of the body around its longitudinal axis, or with the special function of the dentate nucleus (see forthcoming paper by Dr. Clarke and myself).

Should this be so, then its habitual asymmetry would assume more importance in view of Guldberg's interesting and valuable work on the relation between asymmetry and the occurrence of rotatory courses and movements in quadrupedal as well as bipedal animals.

(3) Literature of the Tract.

The tænia pontis was probably first recognised as a definite tract by *Malacarne* [1], who spoke of it as "accessorii d'motori comun" and traced it to the "linguetta laminosa," now known as the lingula cerebelli.

Rolando in his well-known monograph [2] figures the fibres, and states that he found them in the ox and sheep.

Friedrich Arnold also figured [3] the tract under the name "filamenta pontis lateralia."

Henle [4] was the first systematically to describe the bundle in its macroscopic course and to name it as the "tænia pontis."

He stated, however, that it arises from the cerebellar nuclei (thus according to him it "aus dem Mark-kern des Kleinhirns hervortritt"), and that it loses itself on the mesial surface of the crus.

Retzius [5] in describing the isthmus of the brain, points out that the tænia is very constant in occurrence, though differing in degree of superficial development.

Dejerine and *Mme. Dejerine-Klumpke* [6] describe it as passing from the lateral sulcus of the mesencephalon and winding round the crus cerebri penetrating the groove of the third nerve to enter the pons varolii.

Thus, all authors appear to have regarded the tænia as a cerebello-pontine, *i.e.* cerebello-fugal tract, but I propose to adduce facts which go to prove that this view is the converse of the actual truth.

Further, it is frequently spoken of as being inconstant, *i.e.*, as though often absent. This is only owing to the fact that it is often covered by the sub-pial tissue, and so thickly as to be entirely obscured. It should therefore, as Retzius has said, be regarded as a constant anatomical entity, and not described as an exceptional rarity, or an aberrant portion of the pons.

(4) Origin and Nature of the Fibres of the Tract.

So far the precise origin and destination of the tænia has not been determined.

In an interesting case (B—n. U.C.H., 1905) of bilobate cerebellum recently under my observation where agenesis of the vermis was practically complete, there was a concurrent defect in the development of the pontine region, especially of its frontal third.

In this case also the mesencephalic tectum was very poorly developed, hardly any differentiation existing between the corpora quadrigemina.

All the transverse circumferential tracts were exposed, and the frontal portion of the pons spread out in a thin layer over the feebly developed crura. Under these circumstances the well developed (or exposed) tænia pontis was easily seen

FIG. 4.

Macacus Rhesus. Microphotograph of a horizontal section (Marchi stain) of the side of the fourth ventricle and brain stem. The lesion, which was a vertical cut through the lateral fillet, is seen as the horizontally-directed gap in the lowest part of the section.

Dentate Nucleus
Inferior Peduncle
Superior Peduncle
Flocculus
Uvula
Cerebellar Vermis
Lesion

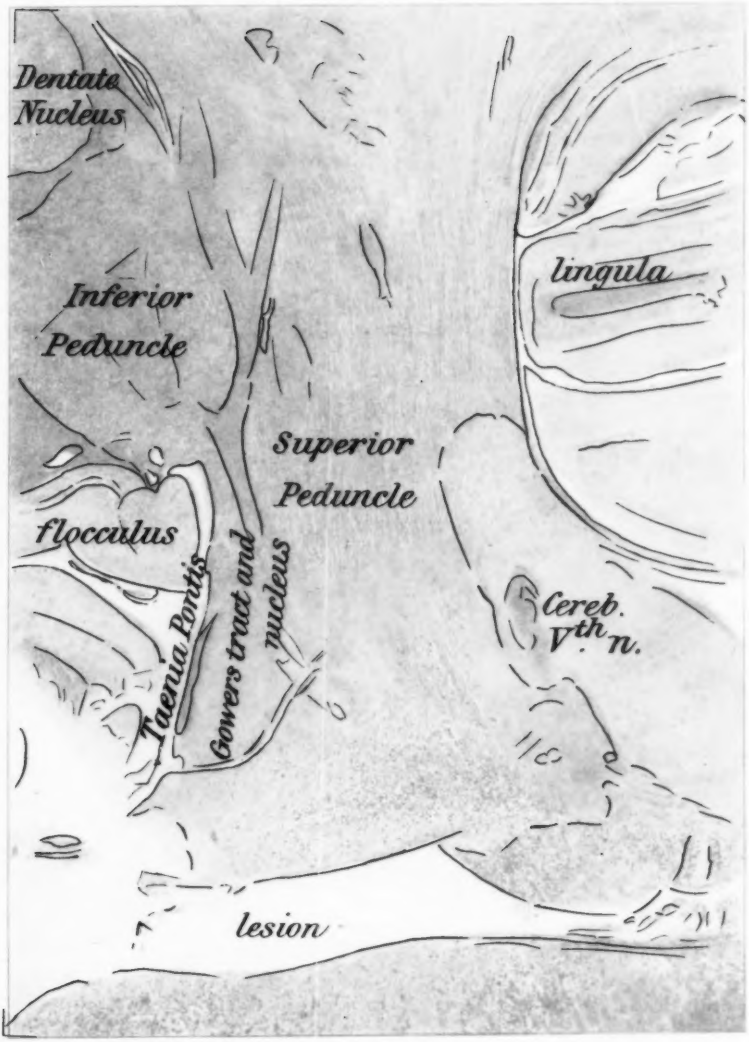


Fig. 4.

Macacus Rhesus. Microphotograph of a horizontal section (Marchi method) of the side of the fourth ventricle and brain stem. The lesion, which was a vertical cut through the lateral fillet, is seen as the horizontally-directed gap in the lowest part of the section.

to take origin contralaterally in a mass of embryonic grey tissue continuous with that of the ganglion interpedunculare but ventral to it.

Cerebellarwards the tænia ran in the normal manner over the lateral fillet to the superior cerebellar peduncle, curved over it and passed beneath the edge of the cortex cerebelli towards the nucleus dentatus which was well developed.

The tænia thus in this case behaved like the other ponto-cerebellar tracts, and as a localised bundle of association fibres, united a portion of the frontal border of the pontine grey matter with that of the cerebellum, apparently the nucleus dentatus (*vide infra*).

In another case of agenesis of the cerebral pallium with bilateral occipital microgyria the tænia was exceptionally well-marked (see fig. 3), and as in the previous case the nuclei of origin and termination were both adequately represented.

As a purely basal tract, the tænia is naturally one which is quite likely to be present, even in cases of considerable failure of growth of the higher centres.

Direction and Termination of the Fibres of the Tract.

I wish now to show that the direction of the fibres of the tænia pontis as estimated by the only means at our present disposal, viz., the Marchi degeneration method, is cerebello-petal like the other ponto cerebellar systems in parallel position.

In a Macacus Rhesus (1904) I divided with the lateral fillet the tænia pontis without injuring the neighbouring border of the pons and middle peduncle of the cerebellum.

As far as can be seen the whole of the fibres of the tænia in this case degenerated cerebello-petally, and as seen in the micro-photograph (fig. 4) and the (Edinger) drawings (figs. 5 and 6) ran up and along the mesencephalon, coursing over the remnant of the lateral fillet and along the lower border of the superior cerebellar peduncle.

As the tract lies on the lateral fillet, it is separated from it by a thin layer of grey substance, which is really continuous with that surrounding Gowers' bundle. The fibres

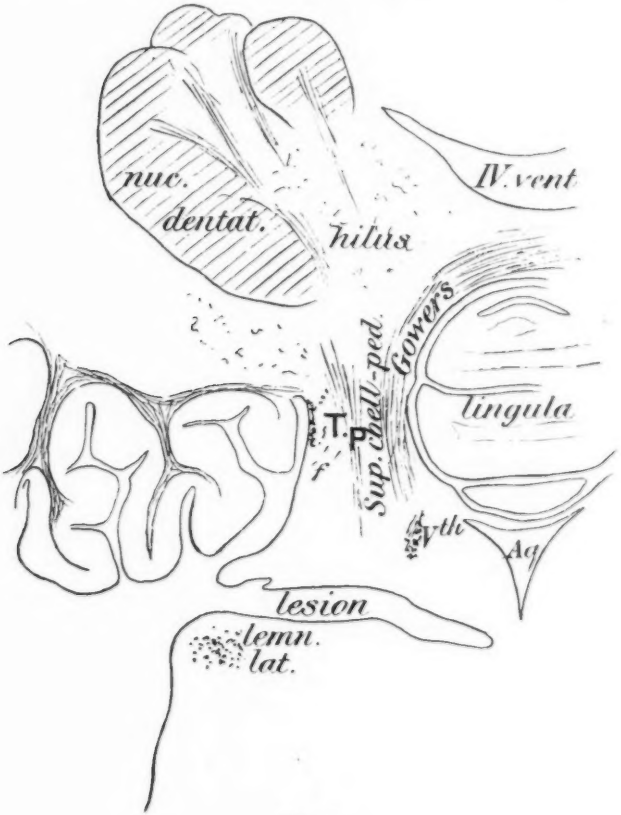


FIG. 5.

Section (higher) through the lower third of the anterior cerebellar commissure. The degenerate tænia is seen to left of letter **T**. A few fibres marked *f* are already leaving it to pass in and through the superior peduncle **P**.

cross the superior peduncle close to the edge of the cerebellar cortex, and thus reach the ventral or frontal commencement of the primary fissure of the cerebellum.

FIG. 6.

Section (higher) at level of decussation of fourth nerve. The tænia **T. P.** is now breaking up and its fibres *f* passing inwards to reach the nucleus dentatus and fastigii.

When it is thus behind the edge of the cerebellar cortex, the tract is a bundle triangular in section which soon spreads laterally while closely applied to the cortex cerebelli of the innermost leaf of the flocculus.

Behind the fasciculus is the mass of the inferior peduncle turning up in front of the frontal edge of the outer shell of the dentate nucleus. The majority of the fibres of the tænia gradually merge themselves between the fibres of the peduncle passing towards the nucleus dentatus in which they appear to end, while a few pass inwards to terminate in the roof nucleus.

A certain loss of fibres was noticeable while the tract was near the cerebellar cortex, but contrary to expectation (in view of the homology between this tract and the mass of ponto-cerebellar fibres) none could be definitely traced into the granular layer of the neighbouring folium.

The termination of the tract in the nucleus dentatus probably explains why Henle with coarser methods of investigation regarded the fibres as coming from that nucleus, at any rate it is evident that he observed a certain relationship between the tract, and what is in this paper asserted to be its real termination.

Literature.

[1] Malacarne (Quoted by Rolando), "Neuroencefalotomia," p. 171.

[2] Rolando "Recherches Anatomiques sur la Moelle alongée." *Memorie della Reale Accademia della Scienze di Torino.* Vol. xxix. Read December 29, 1822.

[3] Arnold "Tabulæ Anatomicæ." Tafel IV., fig. 3, Fasc. 1, 1838.

[4] Henle "Handbuch der Systematischen Anatomie des Menschen." 1871. Bd. III., Abth. 11, S. 129.

[5] Retzius "Das Menschen Hirn," 1896, S. 49; also "Biologische Untersuchungen." 1898. N. F. viii., S. 65.

[6] Dejerine et Klumpke "Anatomie des Centres Nerveux." Paris, 1895, T. 1, p. 327, T. 2, p. 483.

THE MENTAL SYMPTOMS OF CEREBRAL TUMOUR.[1]

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THE older writers upon the mental symptoms of tumour of the brain made various estimates of their relative frequency. Andral [1] in 43 cases, and Durand-Fardel [8] in 70 cases, found that such symptoms were very rare, and thought that they should be regarded as exceptional. Nasse [20], however, found mental symptoms in 19 out of 50 cases, Friedreich [9] in 19 out of 44, and Lebert [17] in 29 out of 90, while Ladame [16], in 1865, noted them in 112 out of 331 collected cases—a proportion of from one-third to two-fifths. Calmeil made the curious observation that, while mental disturbances occurred in over one-half the cases of cancer of the brain, they were rare in tubercle, an hypothesis which subsequent investigations have not confirmed. Some years later Bernhardt [2] found that in more than half of the tumours of the cortex and lobes there were mental symptoms.

In later years the frequency with which mental symptoms occur is regarded as somewhat greater. Giannelli [10] found them in 323 out of 588 collected cases, but he reckoned all cases with speech disturbance among the mentally disturbed. Schuster [25] estimated that fifty to sixty per cent. of all cases showed some such disturbance. Bramwell [5] believes that the great majority of cases show mental symptoms, and Bruns [6] holds that only rarely, when the growth is very small or has some special situation, is mental disturbance

[1] Read at the thirty-first annual meeting of the American Neurological Association at Philadelphia, June 3, 1905.

entirely absent. In the last edition of his "Lehrbuch" [21], Oppenheim says that we may consider it the rule that the sensorium is not unaffected at the height of the disease; a somewhat more liberal estimate than is indicated in his monograph [22] in Nothnagel's system.

It is possible that some of the earlier estimates, like Andral's, were due to the failure to lay sufficient stress upon the minor mental disturbances. Attempts have actually been made to estimate the frequency of mental disturbances by an enquiry into the frequency with which tumours are found at the autopsies of the insane. Fischer found a new growth only once in 318 autopsies, but Leubuscher found a growth four times in 350 autopsies, and Blackburn [4] 29 times in 1,642 autopsies, which is not quite as frequently as I have found tumours in autopsies of patients not insane (101 in 5,069 autopsies at the Boston City Hospital).

I have made an attempt to estimate the frequency of mental symptoms, in cases of tumour of the brain, from a study of the clinical history of 104 cases in which a growth of some nature was found at the autopsy. All but two of these cases are taken from the records of the Boston City Hospital. Many of them came under my own observation, and 39 of them have already been reported in a monograph published some years ago [11]. For the rest, I am indebted to the kindness of my colleagues at the hospital for the privilege of citing them here.

In these 104 cases, mental disturbances sufficient to attract attention were noted in 79, a trifle over 75 per cent. of the cases. On further study, however, and on applying a rigorous criticism, a large number will have to be excluded. In no less than 14 cases the tumour, although of considerable size, was but one manifestation of tuberculosis of the brain, and tubercular meningitis co-existed. In 6 cases there was marked alcoholism; in other cases the new growth was congenital (chordoma) or insignificant, or there were complicating diseases, Bright's disease, typhoid fever, and the like, or the patient was brought to the hospital unconscious, and no history of the previous condition could be obtained. Such cases, 40 in all, have been excluded, even though it

seemed probable that the symptoms of tumour and the symptoms of intercurrent disease might fairly be differentiated.

TABLE I.

	Grand total.	With mental symptoms.	Whole number considered.	With mental symptoms.	With early mental symptoms.
Frontal	8	6	6	5	3
Central	8	7	4	4	2
Parietal	2	2	2	2	1
Occipital	4	3	2	2	1
Temporal	7	7	6	6	5
Corpus callosum	4	4	3	3	3
Optico-striate	17	13	9	9	4
Corpora quadrigemina and pineal gland	3	3	3	3	3
Pons	6	4	1	1	0
Cerebellum	10	7	7	6	1
Hypophysis	3	2	2	2	1
Base, anterior fossa	5	5	5	5	2
Base, middle fossa	1	1	1	1	0
Base, posterior fossa	2	1	1	0	0
Multiple	23	13	11	8	2
Not stated	1	1	1	1	0
Total	104	79	64	58	28

In the 64 cases remaining, mental symptoms were noted 58 times, a trifle over 90 per cent. (Table I.) In considering these figures, many things must be borne in mind. The patients were hospital cases, often ignorant and stupid, often of foreign birth, with no friends or relatives at hand to give an intelligent history of their previous condition. Under such circumstances it is by no means easy to determine whether there has been any change in the mental condition, especially when the mental level is normally low. Furthermore, we must admit regretfully that hospital records are often defective, and that the house officers sometimes fail to report, or even to note, important symptoms. The patients, moreover, came under the care of different physicians, and sometimes were seen only cursorily, if at all, by the neurologists of the hospital. Finally, we lack a definite and generally-accepted method of examination of the mental condition of the average patient, such as we have for deter-

mining sensation, motion, or reflex activity. It would, therefore, not be surprising if the slighter mental changes should sometimes escape observation. It is surprising to me, however, that under such adverse conditions mental disturbances should be so frequently recorded, and it goes far to strengthen the opinion I expressed some fourteen or fifteen years ago [12], which Oppenheim [23] possibly regarded as extreme, "that in every case some change can be found by a competent observer who has known the patient intimately before; in other words, that there can be no gross lesion in the brain without some disturbance, greater or less, in the psychical functions."

A case under my observation a few months ago will illustrate this point. The symptoms pointed very definitely to a tumour in the occipital lobe, but no autopsy could be obtained. The patient was a young man of unusual mental attainments, who had won high honours at two great universities. He was of a high-strung, intense, poetical nature. On many occasions it seemed as if there were no mental impairment whatever, but closer study revealed almost invariably a readily-induced exhaustion of the brain, so great at times as to compel a cessation of enquiry, or even of ordinary conversation. With this was associated an exaggerated emotional instability and a good deal of mental depression. The latter, however, might be regarded as natural under the depressing conditions. He had a quadrant hemianopsia, but under ordinary conditions there was no sign of any psychical blindness. Under the excitement, however, of an examination by a strange physician, he was temporarily unable to give the names of ordinary objects exposed to his view. Yet later, when free from excitement, he could do so normally.

Mental disturbance, as the result of a cerebral lesion, is what we might fairly expect *a priori* from our knowledge of the anatomy and functions of the brain. Anatomically we know that the brain is a mass of inter-connected neurones, each one of which is in contiguity with many others, and may be aroused through many different chains of neurones. If even a small group of neurones be obliterated by disease,

there are some lines of nervous conduction in which a link is broken, and the mental processes become just so far defective or slow in operation. If the single factor of its characteristic smell be eliminated from our concept of an orange, our mental patrimony becomes just so much the poorer. "Any destructive focal lesion of the brain," says Bianchi [3], in his recent admirable treatise on psychiatry, "must also have as an effect a diminution of the mental patrimony and of the vigour of the personality in some direction." With small lesions, however, especially if located in certain regions of the brain, such disturbance might be insignificant, and might not be detected by our present methods of clinical research.

We must also recognise the fact that there are certain growths which are apparently unattended with any cerebral symptoms whatsoever. The chordoma, one of which is included in my series, is a congenital growth, and, apparently gives rise to no symptoms in the few recorded cases where it has been found. Osteophytes in the skull and psammomata may sometimes attain considerable size without giving rise to any disturbance; the same is also true of the small cysts which develop in the choroid plexus. Furthermore, we occasionally meet with growths which are enclosed in a calcified capsule where no history of cerebral trouble can be elicited. Nine of the cases in the present series may be classed under one or the other of these various types, and may fairly be excluded from consideration. They all entered the hospital for some other disease, the symptoms were not cerebral, and the tumour was discovered only at the autopsy. Various cases are on record where the presence of a large tumour was not suspected during life, and where the cerebral symptoms were absent or insignificant. In the present series of cases, however, there were no instances where a large growth existed without cerebral symptoms, although there was often a question whether those symptoms were due to the growth or to intercurrent disease. The statement which I have made above, therefore, needs a little modification to this effect, that in every case of brain tumour presenting any cerebral symptoms some mental symptoms

can probably be discovered—at least, by a competent observer who has known the patient intimately before.

The cases here under consideration, however, were, of course, seen in the terminal stages of the disease a few weeks, or sometimes a few days, before death. One of them, which only recently came to autopsy, came under my personal observation over two years before. At that time the mental symptoms were insignificant, but in the last year of his life, after he had been trephined, they developed to such an extent that he became suspicious, deluded and violent, developed hallucinations of sight and hearing, and had to be committed to an asylum for the insane. Later he grew quieter, and for some months before death was dull and stupid, gradually becoming comatose in the weeks preceding death. In this case the optic nerve presented analogous conditions. So long as he was under my observation no neuritis could be discovered, but after he had been trephined, I learned that he became almost totally blind. The mental symptoms were observed comparatively early in the progress of the disease in 28 of the 64 cases. The importance of the early onset of such symptoms, with reference to the location of the growth, can be more advantageously discussed later, when we consider the relation between the symptoms and the seat of the growth.

In regard to the nature of the mental symptoms occasioned by a new growth within the skull, the prevalent opinion has been that expressed by Oppenheim and Bruns, that there is a steadily-progressing stupidity, beginning as somnolence and mental torpor, and eventually going on to stupor and coma. They both recognise the occasional occurrence of other mental conditions, such as melancholia, hallucinatory excitement, simple dementia, delirium, &c., as well as the peculiar form of mental weakness associated with a childish propensity to jest (Witzelsucht, moria, puérilisme), but Oppenheim regards them as rare, and Bruns believes that when specific psychoses occur, they are to be regarded as due to predisposition, and not caused by the new growth. Dupré [7] describes the characteristic mental condition as one of dulness of the intellect, affective torpor, and inertia of the

will, with, occasionally, ambulatory automatism. He lays especial stress upon a state of puerilism manifested by an infantile character of the psychical reactions, with various modifications of the affective tone, depression or excitement, often associated with the childish joviality above mentioned. Giannelli, however, has pointed out clearly that the mental symptoms of tumour of the brain may be varied and manifold, and this opinion has been confirmed by the exhaustive study of Schuster.

Schuster, as a result of a study of 775 collected cases, found that the conditions of simple mental weakness, dulness, sopor and dementia were present in 423 cases, while 352 cases presented some active symptoms which were classified as follows:—

Symptoms similar to those of general paralysis ...	29
Symptoms similar to those of chronic paranoia ...	19
Symptoms similar to those of melancholia and depressive states	57
Lachrymose with mental weakness	3
Depression with great irritability	1
Mental symptoms as in neurasthenia and hysteria ...	15
Mental symptoms as in mania	13
Euphoria, Witzelsucht, moria, hypomania	23
Irritability, "epileptic character," maniacal and similar states	95
Unrest, anxiety	1
States with confusion and hallucinatory delirium ...	52
States similar to Korsakow's psychosis	2
States similar to moral insanity	7
States with impulsive acts	3
Circular states	5
"Mental weakness with states of excitement" ...	10
State as in senile dementia	1
Unclassified	16

His investigations prove, therefore, that the conditions of stupor and mental torpor, although they are present in a greater proportion of cases, are not in such an overwhelming majority as the statements of Bruns, Oppenheim, and Dupré would imply, and that specific psychoses are sufficiently common to cast some doubt upon Bruns' statements.

My own investigations would agree in the main with those of Schuster, although I have found a lesser proportion of certain states than he has given. The largest number of cases, thirty-one in all, presented the recognised type of mental failure and dulness. The patients exhibited various degrees of languor, somnolence, dulness, apathy, mental torpor, failure of memory, and a general failure of all the mental functions, ending usually in complete stupor and coma. In the earlier stages it was sometimes noticeable, as has been observed by so many other writers, that, when aroused by sufficiently powerful stimuli, the mental processes could be performed clearly and correctly, but with unusual slowness.

Seven cases showed noticeable mental confusion and disorientation, with mental failure, failure of memory, irrelevancy in speech, mild mental wandering, somnolence, stupidity, and a dazed mental condition.

In fifteen cases the mental impairment went on to actual states of delirium, which became wildly maniacal in three patients, and which demanded restraint in several others. Actual hallucinations were occasionally noted. In these delirious states there was also mental failure, confusion, disorientation, failure of memory, and often more or less stupor, mental slowness, incoherence, and vague and unsystematised delusions.

Neurasthenic and hysterical states were occasionally noted in the earlier stages of the disease, but, with a single exception, there eventually developed more marked mental disturbances, so that the cases were eventually classified under one or another of the divisions given above. The diagnosis of hysteria or neurasthenia was, indeed, occasionally made at first by other physicians, although not quite as frequently as I have known it to be made in private practice. One or two patients, who afterwards became much demented, at first showed the scrupulousness and questionings so common in the neurasthenic conditions, while others showed the emotional instability of hysteria.

Vague and unsystematised delusions were occasionally noted in the confusional and delirious states, but in only two

cases were there definite delusions of persecution of a somewhat systematic character. One of these had also well-marked hallucinations of sight and hearing. Only one case presented symptoms of marked mental depression, unattended by any mental failure or confusion. The normal depression, natural upon the appreciation of the disease, must, of course, be omitted from this category. One patient also, whose case I reported some years ago [13], presented a fairly typical picture of general paralysis. The condition of childishness, *puérilisme*, emphasised by Dupré, was noted exceptionally, but true Witzelsucht was not observed. I have seen it in one or two cases of probable tumour in private practice, but in my experience it is a rare phenomenon in cases of brain tumour. It is, however, not uncommon in cases of incipient general paralysis.

I found no very striking differences as to the period of the disease at which the different types of mental disturbance developed. The mental symptoms were of early onset in about half of all the cases, 28 in 58, and they were also of early onset in about half of each of the three main groups, stupor (13 in 31), confusion (4 in 7), and delirium (8 in 16). One distinction, however, must be made. Although some mental symptoms appeared early in the course of the cases which became delirious, the delirium was usually a later feature. Confusion, mental dulness, somnolence and the like, characterised the earlier stages; delirium, violence, and profound stupor the later stages.

Turning now to the question of the relation between the seat of the tumour and the mental symptoms, it may be well first to deal with two general questions in regard to the relative position and size of the growth before we consider in detail the exact localisation.

Schuster has found that, in the collected cases of tumour with mental symptoms, the growth was located in a much larger proportion of the cases on the left hand side of the brain, excepting with tumours of the temporal lobe, where the proportions were about equal. In the frontal and occipital regions there were about four tumours on the left to three on the right, while in the central and parietal

regions the left-sided tumours were about twice as numerous. This agrees with Phelps's [24] study of brain injuries, in which mental symptoms were much more frequent in injuries of the left frontal lobe than of the right. My own cases do not support these conclusions. Of the cases involving the cerebral lobes, only 11 were on the right and 9 on the left side, and the only one of these 20 cases where no mental symptoms were recorded was on the left side. Taking all the 64 cases, 28 were on the right side and 29 on the left, and of those without recorded mental symptoms, 3 were on the left side and none on the right.

In regard to the size of the tumours, it is obvious that a large growth is likely to give rise to more mental, as well as other, symptoms than a small one, and the resultant symptoms are, of course, apt to be more pronounced. I have arbitrarily divided my cases into three classes, large, where the growth was over two centimetres in diameter; medium, from one to two centimetres; and small, below a centimetre. Following this classification, the cases may be arranged as follows :—

Cases with mental symptoms ...	31 large	... 21 medium	... 5 small
,, without mental symptoms ...	2 ,,	... 3 ,,	... 1 ,,
,, rejected	7 ,,	... 13 ,,	... 20 ,,
Total ...	40 ,,	... 37 ,,	... 26 ,,

In one instance the size of the growth was not stated. In other respects the table gives us no very definite date, and gives no special support to our *a priori* conclusions.

Coming now to the special problem of the relation of the mental symptoms to the special regions of the brain, it may be well first to reproduce the three following tables from Schuster. In the first he gives the scale of frequency with which the different regions are affected by tumours in general, as follows :—

Cerebellum	21·6
Multiple	14·7
Frontal	12·1
Central	12·0
Brain stem and crura	9·9
Basal ganglia and ventricles	7·0

Occipital	4·3
Temporal	3·9
Hypophysis	3·8
Medulla oblongata	3·4
Parietal	3·3
Corpora quadrigemina and parietal	3·1
Corpus callosum	2·5

In the second he gives the relative percentage with which the different regions are affected in cases with mental symptoms.

Frontal	18·8
Multiple	18·6
Cerebellar	10·6
Hypophysis	7·0
Basal ganglia, ventricle	6·9
Brain stem, crura	5·9
Temporal	5·8
Central	5·2
Corpora quadrigemina, pineal	5·0
Occipital	4·3
Corpus callosum	4·0
Parietal	2·4
Medulla oblongata	2·3

In the last he shows the percentage of mental disturbances in 100 cases in any given region.

Corpus callosum	100·0
Frontal	79·3
Temporal	66·6
Hypophysis	65·3
Occipital	60·0
Multiple	59·6
Corpora quadrigemina, pineal	53·8
Parietal	52·1
Basal ganglia, ventricle	50·0
Cerebellum	35·5
Central	28·8
Brain stem, crura	25·0

Schuster, moreover, is inclined to the belief that the type of mental disturbance is dependent to some degree, at any rate, upon the location of the growth. The more active

mental disturbances are more frequent in tumours of the frontal, temporal and occipital lobes; the forms of simple mental failure in callosal and parietal tumours. Cases presenting the symptoms of moral insanity and general paralysis are commoner with frontal tumours; cases of the neurasthenic, hysterical or hypochondriacal type with frontal or parietal tumours; while cases of the type of paranoia and mania are rare in frontal tumours; and cases of confusion and delirium occur less often in frontal tumours than elsewhere, and are especially common in occipital growths. He agrees with other recent authors in the opinion that Witzelsucht occurs in tumours of any region, not exclusively in frontal tumours, and believes it is dependent somewhat upon the size of the growth.

Giannelli, drawing conclusions very probably from many of the same cases as those collected by Schuster, agrees with him in assigning special importance to tumours of the frontal lobe and corpus callosum in the production of mental symptoms. His conclusions are as follows: (1) Hallucination, when it exists, indicates an irritation of the corresponding cortical sensory centre, although we can not affirm with absolute certainty that the neoplasm is in the immediate vicinity of that centre; (2) the more the mental disorders—torpor, intellectual arrest, weakness of memory—are manifested at the beginning of the morbid process, the more likelihood there is that the neoplasm is in the frontal lobe and especially the prefrontal region; (3) tumours of other lobes, or in other parts of the encephalon, exhibit the mental disorders above mentioned later in the course of the disease; but, when the mental disorders do appear, they do not differ materially from those manifested by tumours of the frontal lobe, excepting that tumours in the speech area may produce the form of dementia characteristic of aphasic disturbances; (4) these mental disorders indicate a more or less intense and more or less diffuse alteration of the cortical morphological elements; (5) tumours of the corpus callosum are always (?) accompanied by mental disturbances; (6) the modifications of sentiment which may develop in the course of a cerebral tumour have no localising value; (7)

when a cerebral tumour presents the syndrome of general paralysis, it is very probably in the frontal lobe; (8) When ideas of grandeur develop in the course of a cerebral tumour, the tumour is probably in the frontal lobe; (9) Witzelsucht is indicative of a tumour in the frontal lobe, probably on the right side; (10) marked alterations of character, with irritability, violence, insolence, obscenity, threats, indicate a frontal growth; (11) when, in the course of a cerebral tumour, there are exhibited disordered actions of a higher order (forced actions with consciousness) the growth is most probably near the psycho-motor zone, and preferably in the frontal lobe.

In spite of the importance ascribed to tumours of the frontal lobe in the production of mental symptoms, by both Giannelli and Schuster, and in spite of the corroborative evidence, both on the clinical side and from animal experimentation, Müller [19] has contested the generally-accepted opinion that the frontal lobes are associated with the higher psychical functions. Although Müller's arguments are somewhat plausible, I do not think that he has succeeded in controverting the great mass of evidence that has been collected which ascribes to the frontal lobes an important part in the mental processes, although they are in no sense to be regarded as the sole psychical centres. The integrity of the mind must depend upon the harmonious functioning of many regions of the cerebral cortex, and the analysis of even a simple concept will almost invariably show that it is the result of the combination of many sensory processes which demand unbroken links of succession between several widely separated sensory regions. The occipito-temporal region, and especially the zone of language, must play an extremely important part in our ideational processes, and the probability that the corpus callosum is a huge mass of association fibres renders its integrity also important. It is readily conceivable, assuming that a given association tract with a definite function runs from the occipital lobe through the corpus callosum to the opposite frontal lobe, that a lesion in any one of the three regions might break the conduction and give rise to the same phenomena of deficit, just as a lesion

anywhere from the cuneus to the chiasma may give rise to the same symptom of deficit—hemianopsia. It is not unlikely that disturbances in the higher visual centres in the occipital region in Flechsig's posterior association centre, may be responsible for much of the disorientation of confusional states, for example, and that the special phenomena of attention, and many of the ideas of personality, are depen-

TABLE II.

	Total.	Total with mental symptoms.	Dull, stupor.	Confused.	Delirious.	Paranoid.	General paralysis.	Melancholia.	Neurasthenia.
Frontal	6	5	5	—	—	—	—	—	—
	—	iii.	iii.	—	—	—	—	—	—
Central	4	4	2	—	1	1	—	—	—
	—	ii.	ii.	—	—	—	—	—	—
Parietal	2	2	1	—	—	—	—	—	1
	—	i.	—	—	—	—	—	—	i.
Occipital	2	2	1	—	1	—	—	—	—
	—	i.	i.	—	—	—	—	—	—
Temporal	6	6	2	2	1	—	1	—	—
	—	v.	i.	ii.	i.	—	i.	—	—
Corpus Callosum	3	3	—	—	3	—	—	—	—
	—	iii.	—	—	iii.	—	—	—	—
Optico-striate...	9	9	7	—	2	—	—	—	—
	—	iv.	ii.	—	ii.	—	—	—	—
Corpora quadrigemina and pineal gland	3	3	2	—	—	1	—	—	—
	—	iii.	ii.	—	—	i.	—	—	—
Pons	1	1	1	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—
Cerebellum	7	6	5	—	1	—	—	—	—
	—	i.	i.	—	—	—	—	—	—
Hypophysis	2	2	—	1	1	—	—	—	—
	—	i.	—	i.	—	—	—	—	—
Base, anterior fossa	5	5	1	3	1	—	—	—	—
	—	ii.	i.	—	i.	—	—	—	—
Base, middle fossa	1	1	—	—	1	—	—	—	—
	—	—	—	—	—	—	—	—	—
Base, posterior fossa ...	1	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—
Multiple	10	7	3	1	3	—	—	1	—
	—	ii.	—	i.	i.	—	—	—	—
Not stated	1	1	1	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—
Total	64	58	31	7	15	2	1	1	1
	—	xxviii.	xiii.	iv.	viii.	i.	i.	—	i.

The number of cases with early mental symptoms is given in roman numerals.

dent upon associated processes in the frontal lobes; but this is still undetermined, and the cases at present under consideration unfortunately give no definite information upon the subject.

Coming now to the cases that I have collected, and considering them in detail (Table II.), five of the six cases of tumour of the frontal lobe, or, to speak more accurately, of the prefrontal portion thereof, exhibited signs of mental disturbance. In all cases this disturbance was simple mental failure with marked dulness, but in only three of the cases was it noted as being among the early symptoms. The only case where no mental symptoms were noted was a sarcoma, the size of an English walnut, at the foot of the left second frontal convolution near the precentral sulcus, and has already been reported [14]. It is curious that this is the only case of tumour of the prosencephalon or diencephalon in which no mental symptoms were recorded, but mental dulness may readily have been overlooked, as the patient was in bed for a number of weeks, complaining constantly of intense headache, and therefore being averse to any attempts at conversation.

Of four tumours in the Rolandic region, two presented symptoms of mental dulness early in their course, a third, later in the disease, was noisy and delirious, and the fourth, to which I have already referred, finally developed hallucinations and delusions of persecution.

One of the two parietal cases presented neurasthenic symptoms early in the disease. The other became dull and stupid toward the close.

There were six temporal tumours. Two of these were mentally dull, two confused, one delirious, and one presented a typical picture of general paralysis. One of the dull cases did not show much mental change until late in the disease. In all the rest the mental symptoms were of early occurrence.

Every one of the three callosal tumours showed mental symptoms early in the disease, and all became confused, delirious, violent, and demented. One case had hallucinations.

under observation a very short time, one dying suddenly with cysticerci in the brain, the other dying suddenly of surgical shock. A third case, with multiple gliomata in the basal ganglia, has already been reported [15].

In one other case with mental failure the autopsy record of the findings in the brain was lost, the only note being "glioma of brain."

Since mental symptoms were so frequent, no matter in what part of the brain the tumour was located, and since the failure to note such symptoms in a few cases is susceptible, for the most part, of explanation on other grounds than by the assumption that mental symptoms did not exist, the series of cases here presented can hardly give much support to any theory which seeks to establish one special psychical centre in the brain. If, however, we study the cases according to the period of development of the mental symptoms, it may perhaps be of greater advantage.

If we note the early development of the mental symptoms as shown in the final column of Table I., the relative importance of the different regions of the brain in regard to psychical functions becomes more pronounced. In every case of tumour of the corpus callosum or tumour of the corpora quadrigemina, the mental symptoms were of early development. This is in accordance with the other data which indicate that callosal tumours are most frequently attended with mental symptoms on account of the important part played by the corpus callosum in the processes of association. Growths involving the region of the corpora quadrigemina and pineal gland are usually attended with marked dilatation of the lateral ventricles on account of the blocking of the *iter a tertio ad quartum ventriculum*. They thus give rise early to a marked increase of intracranial pressure which might favour the early onset of mental disturbances. Next in importance to these regions in favouring the early onset of mental symptoms come the temporal and frontal lobes. A study of these cases, in fact, has led me to suspect that the temporal lobe, apart from its association with the function of speech has perhaps as important a share in the psychical functions of the brain as the frontal lobe. The other

Seven out of nine cases of a growth in the optico-striate region were mentally dull, but only two showed this symptom early. Two other cases showed early mental symptoms, and both became delirious, requiring restraint, one being wildly maniacal.

Two cases of tumour of the corpora quadrigemina became dull early, and the third also showed early mental impairment, with delusions of persecution. One of them manifested early various neurasthenic symptoms, scrupulosity, and a sense of unworthiness, later growing demented.

The only case of tumour of the pons was mentally dull.

Five out of seven cerebellar tumours were mentally dull, but only one showed this mental failure early. The sixth was delirious. In the seventh case no mental symptoms were noted, but she had headaches with intense vertigo, aggravated by any effort, so that mental symptoms may well have been overlooked.

One tumour of the hypopysis showed mental confusion early in the disease. The second was delirious toward the close.

Of the seven tumours at the base of the brain, five were in the anterior fossa. Of these, one became dull early, three showed mental confusion later in the disease, and a fifth was delirious, showing mental disturbances early. One tumour in the middle fossa became delirious later in the disease. In only one case of a tumour in the posterior fossa, a growth of the cerebello-pontine angle, not involving the brain deeply, there were no noteworthy mental symptoms.

Eleven tumours were classed as multiple, but two other cases might fairly be regarded as in this class, one a multiple growth in the opticostriate region, the other a multiple growth in the pons. They have, however, already been considered under the other headings as the growths were close together. Three of these eleven cases showed mental failure late in the disease. One was confused early, and three were delirious. One of them showed mental disturbances early. One was mentally depressed. Of the three cases where no mental symptoms were recorded, two were

one of those centres should disturb many groups of associated neurones and lead to more diffuse psychical disturbances. In studying the early development of mental symptoms, I have already indicated the relative importance of certain areas in the possible production of such symptoms, but the fact that mental symptoms may arise from a growth anywhere within the cranium forces upon us the conclusion that other factors beside the location are of importance in their production.

Vigouroux [26] has maintained that there is no special region of the brain which elaborates intelligence, but that the intellectual function requires the co-operation of all parts. He thinks that a diffuse alteration of the cortex is requisite to produce obnubilation, confusion and dementia, and that when these symptoms occur they are due not to the growth alone, but to compression, chronic meningitis, and the formation of toxines. Giannelli, indeed, has demonstrated the existence of these diffuse changes in a number of cases of tumour, finding changes in the cells and disappearance of tangential fibres in all parts of the brain.

The factor of pressure is undoubtedly of great importance. We know that pressure will inevitably lead to inhibition of all the cerebral functions. Levassort [18] has tried to show that the habitual mental symptoms of tumour are obnubilation, confusion and weakening of the intelligence, and that these are due to compression of the brain. Outbursts of delirium with hallucinations are exceptional, and are due to toxic influences. Systematized delusions are due to co-existing degeneracy and not to the tumour at all. This seems somewhat strained. Increased pressure existed in many of my cases and was an important factor, but not the only factor. I have already referred to a case with hallucinations of sight and hearing and delusions of persecution. When he first came under observation he had typical Jacksonian epilepsy with slight paresis of the right hand due to an endothelioma growing from the dura in the left mid-Rolandic region. At that time he had no optic neuritis, no noticeable mental disturbances and very little headache. He passed from my observation, but nine months later he was

regions of the prosencephalon, with the opticostriate region and the hypophysis, come next, and the brain stem plays a less important part.

The pathogenesis of the mental symptoms in cases of cerebral tumour is susceptible of various explanations. The mental disturbance may be regarded as a focal symptom, either of deficit or irritation, due to the destruction or irritation of certain centres which have especial psychical functions ; or it may be a general symptom, due either to the increased intracranial pressure, or to the formation of certain toxines in the brain, either from the new growth itself or by the disturbance in circulation caused by the growth.

From a study of these cases we have found that the most common psychical states are those of confusion, delirium and mental stupor or dementia. These states often co-exist, or at least are often present in successive stages of the same case, in mental diseases due to causes other than tumour. This is especially true of the toxic and infectious psychoses, or of the various mental affections which Bianchi has brought under the one comprehensive heading of sensory phrenosis. Persistent hallucinations, obsessions, phobias and limited systematized delusions with a clear sensorium and fairly active intellectual processes were notably rare. It is fair to assume that these later syndromes are dependent upon some derangement in a definite group of association neurones, limited, it may be, in number, but possibly having connections with one another over a wide cortical area. A new growth may disturb some of these growths in its initial stage, but as it increases in size, especially in the case of a growth so large as to involve several cortical regions and the basal ganglia as well, so that its regional classification becomes a problem, the mental symptoms will become more general and widespread. While it seems probable that the functions of certain areas, notably the frontal and temporal lobes, are especially of a complex psychical nature, and that those areas are especially to be considered as psychical association centres, and that the corpus callosum is to be regarded as a vast association tract connecting various association centres, it is not strange if a good-sized lesion in

The cases of diffuse multiple sarcomatosis of the brain, four in number, deserve special mention. One of them entered on account of a huge sarcoma of the neck, which was curetted, the patient dying of shock the same day. No note was made of his mental condition by the surgeons. In two of the others there was marked mental confusion, wandering and delirium; in the third there was simple mental failure.

I have already shown the resemblance between the mental symptoms usually met with in cases of cerebral tumour to those occurring in toxic psychoses. Dupré, an ardent advocate of the toxic origin of these symptoms, has emphasised this point. He believes that the mental disturbances are due to the impregnation of the brain by products of disassimilation, cellular toxines coming from the new growth. He shows that many of the symptoms of tumour of the brain, such as headache, mental disturbance and neuritis, may also arise in toxic conditions, such as uræmia, diabetes, and lead poisoning. He believes that the new growth secretes toxic substances growing at the expense of the brain, and that the gray matter of the brain is peculiarly sensitive to such poisons. He has found, furthermore, lesions throughout the brain, in cases of tumour, similar to those found in toxic-infectious troubles, and calls attention once more to the importance of toxines in the production of optic neuritis. Pressure alone is hardly capable of producing optic neuritis, but, when pressure is combined with the influence of some toxine, neuritis is speedily produced. The cases under consideration reveal nothing as to the nature, or even the existence, of these toxines, but it seems probable that while the situation of the growth is often of influence in producing mental symptoms, especially in the early stages of the disease, and possibly has an influence upon the nature of the symptoms, a combination of increased cranial pressure and the action of toxines is of greater importance, and in some cases may be the only factor to be considered in the production of such symptoms.

trephined. It was reported that the growth could not be removed on account of hæmorrhage. A hole four centimetres in diameter was left in the skull, and hernia developed. A year later he became blind, the mental symptoms above mentioned developed, and he was sent to an asylum. Later he became extremely dull and stupid, dying in coma. The mental symptoms and blindness developed after the pressure had been relieved by trephining.

In considering the possibility of toxic influences, I have tried to see whether the nature of the growth had any influence on the development of mental symptoms. That a rapidly-growing tumour will more speedily cause mental symptoms than one of slow growth is obvious, but beyond that I have been unable to find any special relation between the nature of the growth and the form of mental disturbance, except that delirious conditions were more apt to occur in cases of sarcoma, as the accompanying table will show. (Table III.)

TABLE III.

				Type of Mental Disturbance.						
Nature of growth.	Total cases.	Total considered.	With mental symptoms.	Dull, stupor.	Confused.	Delirious.	Paranoid.	General paralysis.	Neurasthenia.	Melancholia.
Tubercle	21	4	4	2	—	2	—	—	—	—
Glioma	17	14	12	9	2	—	—	1	—	—
Sarcoma	11	11	9	3	1	4	1	—	—	—
Gumma	8	8	8	6	1	1	—	—	—	—
Cyst	5	4	3	1	—	1	—	—	1	—
Endothelioma ...	4	4	4	2	—	1	1	—	—	—
Cholesteatoma ...	3	2	2	1	1	—	—	—	—	—
Carcinoma ...	1	1	1	1	—	—	—	—	—	—
Cysticercus ...	1	1	—	—	—	—	—	—	—	—
Dermoid cyst ...	1	1	1	—	—	1	—	—	—	—
Angioma ...	1	—	—	—	—	—	—	—	—	—
Aneurysm ...	1	—	—	—	—	—	—	—	—	—
Chordoma ...	1	—	—	—	—	—	—	—	—	—
Enchondroma ...	1	—	—	—	—	—	—	—	—	—
Fibroma	1	—	—	—	—	—	—	—	—	—
Osteophytes ...	1	—	—	—	—	—	—	—	—	—
Psammoma ...	1	—	—	—	—	—	—	—	—	—
	79	50	44	25	5	10	2	1	1	—

A STUDY OF THE MINUTE STRUCTURE OF THE OLFACTORY LOBE AND CORNU AMMONIS, AS REVEALED BY THE PSEUDO-VITAL METHOD.

(With Remarks on the Plan of Nervous Structure of Vertebrates in General).

BY JOHN TURNER, M.B.ABERD.

I.

It is now five years since I first published the description of a process of primarily staining the brain in a mixture of methylene-blue and peroxide of hydrogen. At that time, although the merits of the method were evident, its usefulness was largely discounted by the fact that it was extremely untrustworthy, and for this reason I suppose no one except myself has yet worked with it. Another disadvantage was that it did not react with the brains of lower animals.

Since that time I have made numerous experiments towards perfecting the method and rendering it more constant, and these have so far succeeded that I can now generally rely on not only getting a successful result with suitable human brains, but also with those of animals (cow, rat, rabbit, mouse, &c.). And now, indeed, with these animals, especially the rat, the results are more constant than with human brains. For one reason, probably, the conditions are more constant, as with animals one is dealing with presumably normal material, and further, the animal's brain can be kept subjected to a suitable temperature from the moment of its death.

These experiments support the idea, expressed a year ago, that a successful reaction depends in some way upon a development of the *post-mortem* lactic acid fermentation

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in the tissues, but any signs of decomposition, shown by an alkaline reaction of the material, are fatal to success. Even now, in successful cases, the reaction is limited to parts of the tissue. In the case of a small brain, stained entire, I have never succeeded in getting the whole mass to take on the reaction, which in some cases is more diffusely spread than in others, so that parts of the cortex, central nuclei, olfactory lobe and cerebellum will react; in other cases, whilst one small portion is beautifully stained, all the rest is a failure. Spinal cord still remains refractory, although doubtless this part also will ultimately yield to experiments.

The extraordinary amount of detail which the method shows, coupled with the fact that, with the exception of Ehrlich's vital method, it is the only one that I know of which exerts a selective and differential action on the nerve-cells, renders it of such value that I am very anxious that others should give it a trial.

If nervous tissue is placed directly into a watery solution of methylene blue alone, the penetration of the stain is very bad, but, so far as it goes, all the cells are coloured a deep bright blue. The chromatoplasm is the part, *par excellence*, which absorbs the stain. On the addition of peroxide of hydrogen to the blue, not only is the penetration of the stain much facilitated, but also a process of decolorisation of the cells takes place, not, however, extending equally to all of them. To take the case of cortex, whilst the cytoplasm of the pyramidal cells (and I include under this term the orientated cells of all the layers) is almost entirely decolorised, another class, with, amongst other characters, a lack of orientation, resists the decolorisation much longer, so that the cells retain a dark colour. It is to be noticed, also, that the difference of colour between the two classes of cells is not one of degree alone. The dark colour is not a blue of the character of methylene blue, but almost a black, and even when the cells are more decolorised they still have a different tint, as a rule, from the pyramidal cells.

Hence it is possible, by the addition of peroxide of hydrogen to the methylene blue, under suitable conditions,

to obtain a very beautiful and, at the same time, a very valuable differential staining of the nervous system.

We do not meet with abrupt partitions and boundary lines in Nature; these are human conceptions, and therefore the differential action does not absolutely divide one class of cells sharply from the other; sometimes apparently healthy pyramidal cells stain darkly, but this action can to some extent be controlled by the amount of peroxide added to the stain. The fact that sometimes, in the midst of a successful piece, a pyramidal cell and its branches stands out deeply stained is not altogether an objection, as, by this means, details in the relationship of fibrils to nerve-cells are rendered visible, which would not otherwise be so. The differential staining is much truer in the lower animals (*e.g.* rat) than in man. In the former it is not unusual to find places where the division between the "dark" and "pale" cells appears absolutely true. Probably the reason for this is that here one is dealing with healthy tissue, whereas (in my case) the human material is from a morbid source.

II.

The procedure is simple, and can be divided into (*a*) pre-staining, (*b*) staining, and (*c*) fixing stage.

(*a*) *Prestaining.*—The brain is sliced into pieces about 3 mm. thick, and laid on an inverted glass capsule which is placed in a larger receptacle and surrounded by a little distilled water, to which is added from six to eight drops of formalin. The cover of the larger dish is then put on, and the whole placed in the incubator at a temperature of 24° to 25° C., and left for some hours. Numerous experiments show that the best results with rat's brain are obtained after thirty hours, and probably the same applies to rabbits. With mice I have not got quite such good results as from rats, but with these, again, thirty hours yielded the best results. It must be distinctly understood that any signs of decomposition are to be avoided, as under these circumstances the staining will not succeed. Pieces of moist litmus paper placed on the tissue should keep their red colour.

(*b*) *Staining*.—The pieces are then placed in a stoppered bottle in 15 cc. of a 1 per cent. solution of patent methylene blue (Grübler) containing 12 minims of lactic acid to each 100 cc.; 1 cc.[1] of dioxygen added to the stain, and the bottle replaced in the incubator and left four or five days. The subsequent addition of small quantities of dioxygen (0·5 to 1 cc.) on the next and following day seems, as a rule, to promote better penetration of the blue, and also to bring out the fine detail somewhat more clearly. It is well to divide the material into two parts, placing them in different bottles, and varying the procedure as regards the additional dioxygen in the two.

(*c*) *Fixing*.—The pieces are rinsed in water and put into 30 cc. of a freshly made 10 per cent. solution of molybdate of ammonium, to which two drops of hydrochloric acid have been added. They are left in this till next morning, washed under the tap all day, then placed in absolute alcohol, frequently changed, for twelve hours; passed through chloroform, embedded in paraffin, and cut. A drop of xylol is placed on a slide and the section placed carefully on it. They must not be fixed to the slide by Gulland's method, as the contact with water extracts all the finer detail.

The above is the ordinary procedure for obtaining nervous detail, but it may be varied slightly to suit certain parts, *e.g.*, whilst the further addition of 1 or 2 cc. of dioxygen is often advantageous in the case of cerebrum, it does not appear to be so in the case of olfactory bulb. In BRAIN, 1904, I described a modification of the method whereby certain glial detail is shown. In these cases I fixed the fresh tissue by subjecting it to a temperature of 50° to 100° C. for some hours. I find now that in many cases the subsequent addition of dioxygen on two or three occasions, using altogether as much as 2 or 3 cc., results in the staining out of some of the glial elements, notably the mesoglial cells of Ford

[1] Or better still 2 cc. if the tissue will stand it. When too much dioxygen is first added, gas is evolved in the tissue, which becomes spongelike and useless. The pieces should float after a few minutes from the time they are first placed in the staining mixture, but after ten or twelve hours they should sink, and if on the contrary they still float it is due to contained gas bubbles which destroy the integrity of the tissue. Dioxygen is the trade name of a 3 per cent. solution of hydrogen peroxide, prepared by the Oakland Chemical Company, New York.

Robertson, and the Golgi-nets which appear to be derived from them.

In twenty-one animals including mice, rats, kittens and rabbits, only two complete failures were met with. This series included nearly fifty observations, as in many cases the material was divided and different pieces treated in different ways. But although success is now so frequently obtained, the successful parts are still limited, in some cases only the bulb reacts, in others only the cornu ammonis, and so on. And it must be emphasised that unsuccessful early sections should not allow one to condemn the whole block prematurely, as very often on cutting further into it a successful area is lighted upon.

III.

I have chosen to deal with the structure of the olfactory lobe and cornu ammonis, so far as is this is shown by my method, for two reasons: firstly, they are regions about which our knowledge is perhaps most imperfect, and I believe the method I have used shows details which are either not shown by other methods, or not so distinctly; secondly, as regards the cornu ammonis, as it is the oldest phylogenetically, so it appears to me to be not only the most primitive, but in a sense the typical form of cortex, and further, as the cornu ammonis is probably an olfactory type of cortex, it is best studied in conjunction with the olfactory lobes.

The description which follows does not pretend to be exhaustive, as I shall in great measure limit myself to those features shown by my method.

Olfactory Bulb.

In the human subject small patches of gray matter, connected by thin strips of the same, are met with at irregular intervals all along the olfactory track and within the bulb itself. These so-called "islands of gray" present the same features as the cortex from which the tract springs. In the rat and cat an outgrowth of the cortex occupies the portion of the bulb nearest to the cerebrum, and in horizontal sections, it is seen dipping into the olfactory lobe as a triangular

wedge with the apex directed forward. And although its structure is very sharply defined from that of the olfactory lobe, yet sometimes the long dark branches of the darkly stained cells of the pallium are seen wandering out into the substance of the olfactory lobe.

As the glomeruli do not react to the stain, I shall pass them over merely noting that they are situated all round the surface of the lobe, except for a small area in its upper aspect; and that they appear as pale, homogeneous, oval or round bodies, surrounded by several layers of small nerve cells (with nuclei of 4 to 6 μ), some of which are seen sending down stout apical processes into the molecular layer. Passing up between these cells, also, are stout processes from the mitral cells which enter the glomeruli, where sometimes they can be dimly seen breaking up into further sub-divisions.

Molecular layer.—This, like the glomerular layer, is widest on the under-surface, where it obtains a depth of over 0·5 mm., and narrowest on the superior aspect, where it measures only 80 to 90 μ. These measurements are taken from the rat's olfactory bulb. Near the glomeruli are a number of cells (superficial mitral), staining pale blue, regularly arranged, with the apices passing to the glomeruli. In the deeper parts of this region nerve-cells are occasionally seen, but they are few in number. Throughout the layer small, darkly stained, oval or irregularly shaped cells are scattered (12 by 10 μ). They branch very richly, but chiefly in a longitudinally vertical plane, so that in sections cut horizontally or transversely the branches do not appear in any number. Although very delicate, they are of a loose texture, irregular in outline, in parts no thicker than a single neuro-fibril, in others several times as wide, and show numerous little irregular, round or oval swellings, generally only a few microns from each other. These little swellings have also a loose texture, with very generally a pale centre. The branches are extremely sinuous, and on many of them, passing at right angles from the main stem, are little lateral offshoots, which often appear to terminate in a knob or swelling, but sometimes a delicate thread can be traced from the knob running for a

considerable distance as a beaded fibril. These branches pass to the "pale" cells seen in this layer, around and over which they course, forming a pericellular structure, similar to that seen over the pyramidal cells of the pallium in man, but not of such a complicated nature. Appearances strongly suggestive of direct fusion of the branches from

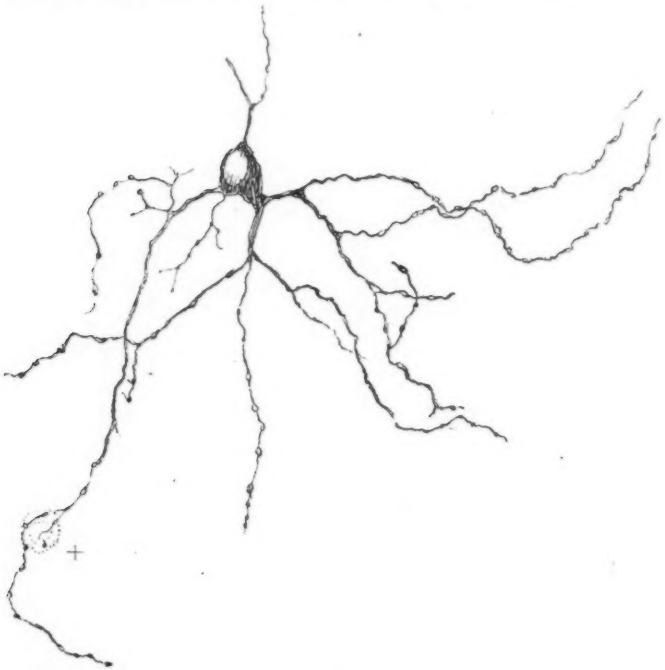

FIG. 1.

Small multi-branched "dark" cell from the molecular layer of rat's olfactory bulb. At + is an appearance suggesting direct union of branches from two cells, and forming part of a pericellular arrangement over the "pale" cell indicated by dotted lines. An occasional lateral short branch, ending apparently in a knob is seen.

different cells are frequently met with (fig. 1). So far I have not been able to identify a definite axon to these cells, either in the rat or cat.

My method shows only very occasionally any traces of the course of the mitral cell branches through this layer,

but besides the sinuously disposed branches just described are others, which, for the most part, run in a nearly direct course from mitral cell layer to glomeruli, and not far apart from each other. They are extremely delicate, best seen in horizontal or transverse sections, stand out sharply, and present along their course, at distances of several microns, comparatively large swellings. These are the prolongations of the olfactory granules (plate i., fig. *A*).

Mitral cell layer.—Although mitral cells are found throughout the molecular layer, yet they especially congregate between the molecular and granular regions, where

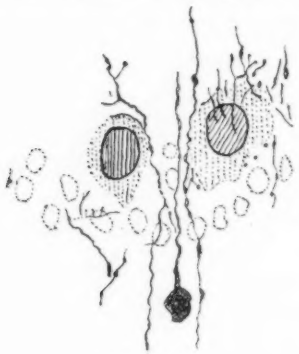

FIG. 2.

Two mitral cells (olfactory bulb of rat) and a darkly stained granule. Other faintly stained granules are indicated by dotted outlines. Shows beaded processes investing the mitral cells.

they form a definite layer, only one cell deep, except on the inferior aspect of the bulb near the mesial line, here the glomeruli are packed up in large numbers like a pyramid with its base downwards; the molecular region is very wide, and on the anterior and posterior aspects of the glomerular cone the mitral cells lie two or three deep without any definite orientation.

They belong to that class of cells which, with the method used, stains of a very pale blue or not at all. In successful preparations the cell body is shown, and it appears as a very pale blue or colourless mass of an oval or triangular shape, about 30 by 15 μ, with a darker, well-defined nucleus, 10 by 7 μ. The main branches pass off

gradually and are stout and colourless, and soon lost to view; in their passage through the molecular layer they branch very slightly. In some cases the chromatoplasm of the cells is visible, consisting of bulky flakes arranged very much as in Purkinje's cells.

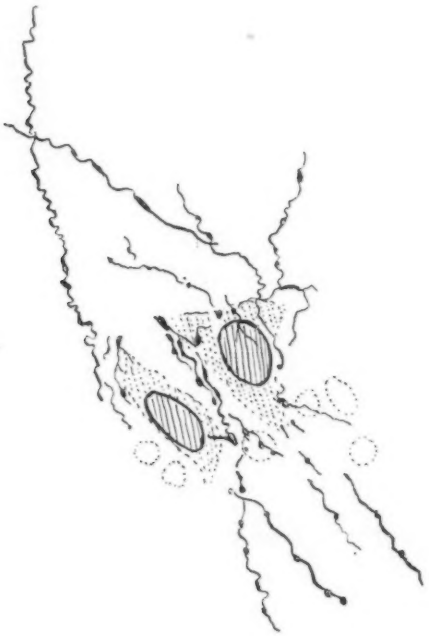

FIG. 3.

Two mitral cells (rat), with investing fibrils which are in great part the continuation of the long apical processes of granules. Some of these are shown passing up through the molecular layer.

The branches of both the granular and the molecular cells (figs. 2, 3, 4) appear to arrange themselves specially around the bodies and processes of the mitral cells, and from the branches of the molecular cells are given off delicate lateral offshoots which pass over or under the same,

producing a pericellular investment similar to that seen about the pyramidal cells, but more rudimentary. That this is not merely an accidental propinquity is shown by the fact that sometimes when the cytoplasm of a mitral cell has somewhat shrunken away from the edge of the matrix, leaving a definite pericellular space, a delicate beaded process may be seen still clinging to the border of the cell and following its outline.

Granule layer.—The granules are disposed in layers of two or three deep, and each layer is broken up into groups. There are from ten to thirteen layers in the rat. The sub-

FIG. 4.

A mitral cell lying in the midst of the molecular layer. By its side is a small "dark" cell giving off branches which divide and invest the body of the mitral cell.

stance intervening between the rows and packets consists largely of fine axis cylinders. Each packet shows a number of pale blue round nuclei, 4 to 5 μ in diameter, around each of which is a thin rim of colourless cytoplasm. Scattered throughout these layers are the granules which have stained darkly. These for the most part are oval bodies with a long apical process, passing straight up through the mitral cell layer, and many of these branches from the more external cells pass completely through the molecular layer. From the other end of the oval a very delicate axon is given off, which in some instances can be traced towards the

FIGS. 5, 6, 7.

Granules (rat), showing apical processes on which, at irregular intervals, are large black oval beads. The very delicate axon is given off from the opposite side of the cell body as a rule, sometimes at right angles from the side, sometimes from a relatively stout basal process, and sometimes after a short course divides again and again (fig. 6). The central part (nucleus) of the granule is generally much paler, and a black thread is often seen passing across it (fig. 5). Note the fusion of the bodies of the granules (figs. 6 and 7).

centre, without branching, for 160 μ. In other instances it very shortly divides and subdivides again (fig. 6). On these axons are very small beads. Sometimes the axon passes off from the side of a granule and runs along in the bundles of fine axons separating the different layers. The nucleus occupies the greater part of the cell body, sometimes it is quite obscured, at others it is seen as a somewhat paler body, bordered by a thin rim of black cytoplasm, which is thicker at each pole of the cell. Very often across the nucleus from apex to base a black line stretches (fig. 5). These cells are generally bipolar, varying from 5 to 8 μ, but occasionally they also possess a very slender lateral branch. The apex is very slender, passing straight up towards the mitral cell layer and exhibiting a series of short, decided twists like a corkscrew, especially near its origin. On it at irregular intervals are very prominent little swellings, which in some cases are nearly 4 μ in long diameter; the larger of these may be further from the cell body than the smaller. I have never seen any lateral projections of the nature of thorns on the processes of these granules. Very frequently two granules appear fused together (fig. 7). The dark granules are very regularly scattered over the field, being most numerous in successful preparations, and in these some of the pale blue nuclei before mentioned begin to take on a deeper colour.

The very great majority of the "dark" cells in the granular layer correspond to those just described, but here and there a larger cell is found (answering probably to the larger granules described by Hill). These latter are of various shapes and range from 12 to 18 μ in diameter. They are more richly branched, and their processes pursue a much more sinuous course and give off little lateral twigs at right angles. I have not been able to trace their axons to the same extent as Hill.

Among the granules, generally a little below the mitral cell-layer, the cells described by Hill as "bracket cells" are met with, but rarely more than one or two in a section. They give off long dendrites on each side, which run

PLATE I.

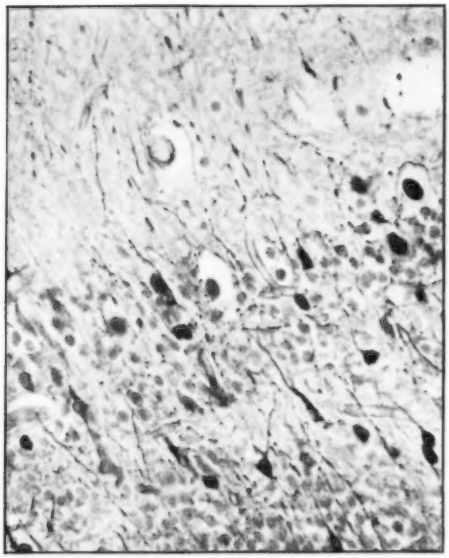

FIG. *A*.

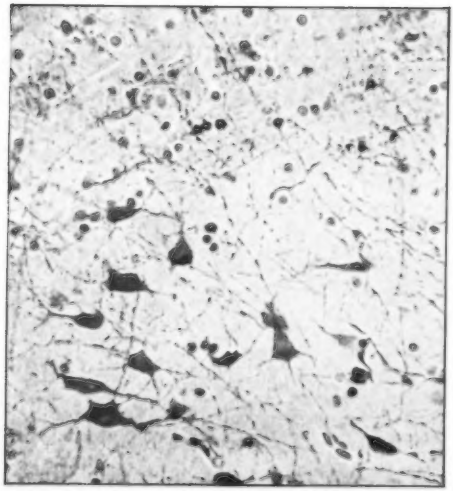

FIG. *B*.

parallel to the mitral cell layer. The branches can sometimes be followed for over 200 μ, they are irregularly moniliform, and apparently give off a few fine lateral twigs near their end. Other large "dark" cells are also met with in comparatively few numbers throughout the granule region.

In the central part below the granules are still larger "dark" cells (39 μ) with numerous long, sinuous processes, on which are long lateral twigs which appear to end in a knob.

In longitudinal vertical sections passing through the anterior commissure the "dark" cells of the pallium send down numerous long beaded fibrils, which run within the commissure at right angles to the course of its axons.

Cornu Ammonis.

In sagittal or horizontal sections, the passage over from the cortex of the pallium to that of the cornu ammonis is sharply defined. The second layer of cells stops at some distance from the beginning of the cornual structure, a little further on the lower layer of cells (third and fourth) narrows down somewhat, and a small strip of matrix intervenes between them and the stratum pyramidalum, but long dark beaded processes stray out from this latter region and pass for considerable distances among the cells of the pallium. In transverse sections the transition appears much more gradual. The following description, unless otherwise stated, is taken from the rat's brain. In either horizontal, longitudinal, or transverse sections the following layers are met with:—

(1) *Alveus.*—This, which represents the medullary centre of the horns, is a thin band consisting for the most part of axis cylinders which may be divided into two layers, the superficial runs more or less tangentially, the deep more or less longitudinally. Its fibres pass on into the fimbria, and so to the hippocampal commissure. An occasional "pale" nerve-cell is met with in the deeper layer. Numerous dark beaded fibrils from the cells of the stratum oriens pass up into this layer, and then bending over run in the direction of the axons (plate i., fig. *B*).

(2) *Stratum oriens* is probably representative of the spindle-cell layer of the cortex. It is characterised by the presence of numerous large (12 by 16 μ) "dark" cells, scattered about in all directions, which give off slender branching dendrites, forming a somewhat coarse-meshed feltwork. Many of these branches, as already mentioned, pass up into the alveus, others run down between cells of the stratum pyramidalum. Exceedingly delicate axons can sometimes be seen, but I have not succeeded in tracing one for any considerable distance. They generally pass in the direction of the alveus. Near the stratum pyramidalum a few "pale" cells are met with, representing apparently displaced pyramids (plate i., fig. *B*).

Stratum pyramidalum.—This layer averages 120 μ in thickness, and consists of three to six rows of pyramidal cells closely packed together with no intervening matrix. The bodies of these cells are from 10 to 18 μ wide and about 18 to 24 long. All the apices pass in one direction through the next layer, where they appear as tolerably wide, straight, colourless strands. In some preparations Golgi-nets can be seen closely investing the nerve-cells. Here and there is a "dark" cell of a similar character to those seen in the stratum oriens. Besides their colour difference, they contrast sharply with the pyramids by their lack of orientation and the abrupt fashion in which their long dark sinuous thin dendrites arise. They are often much larger than the pyramids, some measuring 21 by 21 μ. From these cells, as from those in the stratum oriens, arise the fine beaded fibrils which invest the pyramidal cells. It is no uncommon thing to meet with a "dark" cell lying in this stratum with long thin dendrites, which reach on one side to the alveus and on the other to the stratum lacunosum.

Sections passing through the bodies of the pyramids at right angles to their long diameter show very clearly the general characters of this layer (plate ii., fig. *C*). The pyramids are represented as colourless square, oval, or pentagonal bodies, containing a faintly coloured central nucleus. The apices of these cells are represented as white spaces (but always much more bulky than the dendrites of the

PLATE II.

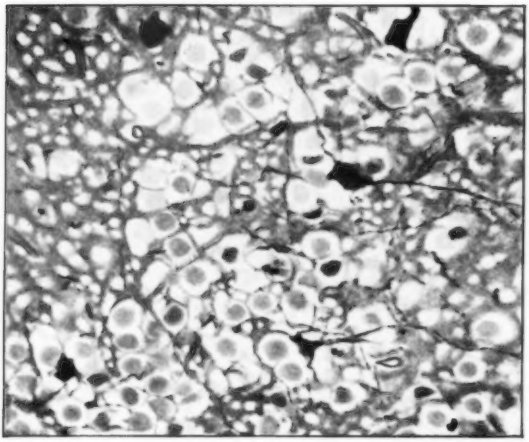

FIG. *C*.

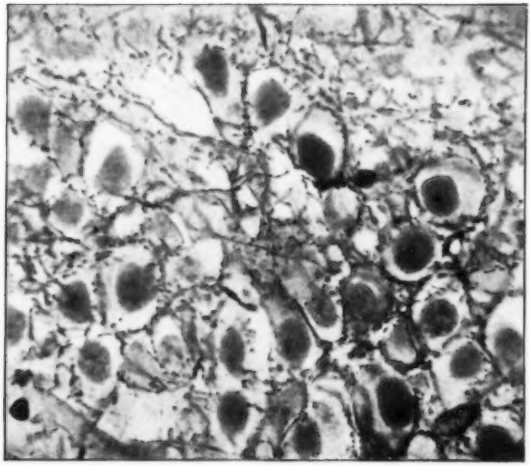

FIG. *D*.

"dark" cells) lying thickly set in a darker matrix (stratum radiatum). Here and there the multipolar cells form a prominent contrast to the pyramids, giving off slender branches which ultimately subdivide into the finest beaded processes.

In some sections from the mouse the apices of the pyramidal cells have stained darkly. The dark colour is not uniform, but broken up by small colourless spaces. It is probably caused by the retention of the stain in the meshes

FIG. 8.

Darkly stained apex of a pyramidal cell from the stratum pyramidalum of man showing mossy or shaggy edges, due to numbers of extremely delicate short lateral fibrils, apparently ending in knobs. Several neuro-fibrils are shown joining on to some of these knobs. The two long fibrils get stouter as they pass away from the apex, indicating that they belong to fibrils passing to join the pyramidal cell, and not that they are given off from the pyramidal cell.

of the Golgi-nets. At the edges of these apices are very delicate short fibrils, passing off at right angles all round the process, thickly set, and terminating in a knob. Some times it seems possible to trace one of these lateral projections beyond the knob as an excessively fine fibril.

Man. — In man, not only are the pyramidal cells relatively more numerous, but they are no longer so closely packed together, and tend to encroach considerably on the stratum radiatum, which is thus in its cell-free portion

relatively much narrower and less distinctly demarcated than in lower animals. Delicate beaded neuro-fibrils course in all directions throughout the matrix between the pyramidal cells, and form a definite investment about the

FIG. 9.

Cortex of cat. A branching neuro-fibril passing to join the apical processes of two ganglion cells, either, it is suggested, to mingle with their intercellular fibrils, or to take part in their pericellular networks.

body and branches of these latter, an investment of greater complexity than in the rat or rabbit. In preparations where occasionally a pyramidal cell and its branches stain darkly, even the finest twigs show closely set lateral knobbed processes, by means of which these branches can be dis-

criminated from the branches of the true "dark" cells, which are devoid of these lateral excrescences. Very often delicate primitive fibrils appear continuous with certain of the lateral processes, as shown in fig. 8. And it should be

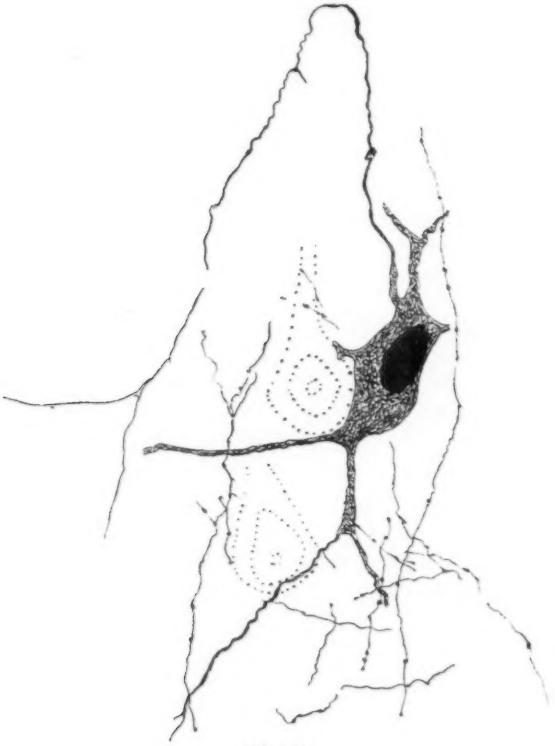

FIG. 10.

"Dark" cell in human stratum pyramidalum, the axon of which at first passes upwards (towards stratum radiatum), and then looping on itself runs down in the opposite direction. Two ganglion cells are shown in dotted outline, and some of the beaded neuro-fibrils of the intercellular plexus.

observed that these fibrils become stouter the further they recede from the pyramidal cell process, a feature which is opposed to the idea that they are an integral part of the pyramidal cell to which they appear to join; in other words,

they do not represent a direct passage out of the intracellular neuro-fibrils, but rather the passage of an extracellular neuro-fibril to the fibrillar investment of the pyramidal cell.

The true "dark" cells of this region are relatively smaller than those found in mouse, rats, and rabbits, their axons may often be detected, frequently passing off in a direction towards the stratum radiatum, but after running for a short course in this direction they generally bend round and run down towards the stratum oriens (fig. 10).

(4) *Stratum radiatum.*—This region is a layer 240 μ thick in its broadest part, but much narrower at the junction of the cornu ammonis with the pallium. It has a radial appearance owing to the presence of the apices of the pyramidal cells, which run straight up through it as relatively stout (2 μ) colourless bands, closely set together and giving off very few side branches. Passing up between them are a number of much more delicate beaded or moniliform branches from the "dark" cells. The moniliform swellings are generally oval and may be 4 μ long. Sometimes they appear as rings having a colourless centre. Very few cells are met with in these layers. Towards the nucleus fasciæ dentatæ, where "pale" and "dark" cells lie jumbled together, long beaded processes cross and recross in all directions.

(5) *Stratum lacunosum.*—This layer probably represents the second layer of the cortex, it is indistinctly marked off from the neighbouring region, is about 70 μ thick, and is characterised by small pale nerve cells of 18 to 20 μ in diameter lying widely separated from one another. In many cases only their nucleus is visible, as the cytoplasm does not stain. An occasional small cell of the dark variety is also met with. The colourless apices of the pyramidal cells are continued on into this region, and probably pass quite through it. The most conspicuous feature is the somewhat loosely meshed feltwork formed by the division and subdivision of the dark beaded branches, which here lose their regularity and cross in all directions. Although no definite pericellular network is seen, yet unquestionably the beaded

PLATE III.

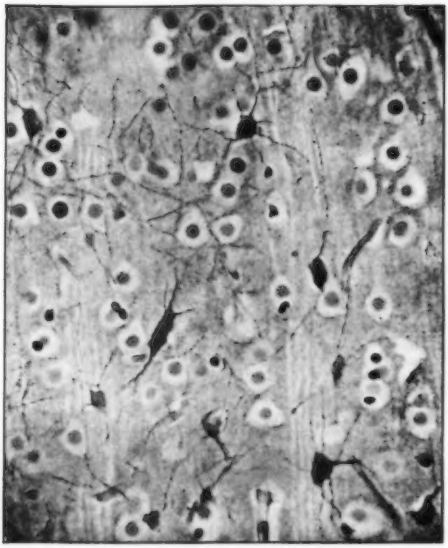

FIG. *E.*

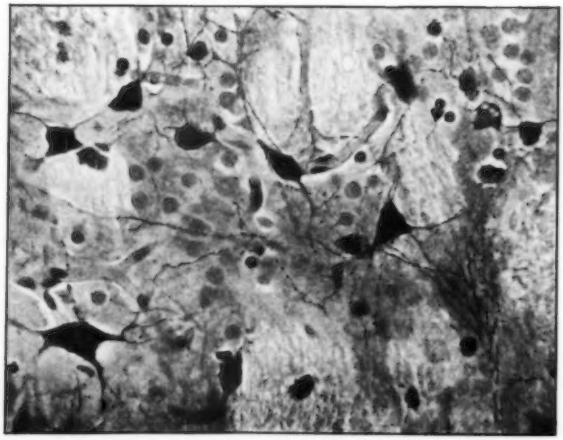

FIG. *F.*

fibrils are more closely set around and about the pale nerve-cells, and they often appear to be directly joined to one another.

(6) *Stratum moleculare.*—On the outer side of the last stratum the beaded fibrils rapidly diminish in number, forming a fairly sharp boundary between it and the stratum moleculare. It is customary in text-books to subdivide this region into two, an inner stratum moleculare and an outer, the nuclear layer (zonal band of first layer). But this subdivision, though it unquestionably exists, is not evident in my preparations, and the whole region seems to be a direct continuation of the first layer of the cortex. It contains a few small "pale" cells, and about an equal number of "dark" ones, from the latter of which delicate branches are given off and ramify somewhat sparsely throughout this layer. Sometimes one of these "dark" cells lies close against the free border of the zone.

This layer is separated from the dentate fascia by a fold of pia mater.

Dentate Fascia.

(7) *Stratum moleculare.*—Quite similar in character to that above described, of which it is the direct continuation, bent round upon itself.

(8) *Stratum granulosum.*—This layer invests the lower and inner portion of the recurved stratum pyramidalum, somewhat in the form of a stocking cap, with its closed end directed towards the posterior aspect of the brain. It consists of a belt of small round or ovoid "pale" cells, closely packed together, their average size being 10 by 8 μ, with a nucleus (6 μ) occupying the greater part of the cell body. They are about the same size and have very much the same characteristics as the cells of the second layer of the cortex. In the region of the subiculum in man, where the cornual structure begins to appear, one of the first changes noticed in the cell architecture is the disappearance of the second layer, which is replaced by little islets of cells separated from each other by the interposition of larger pyramidal cells. In these islets the cells are closely

packed together. Ultimately, when the true cornual structure is well established, they quite disappear, but from topographical and other reasons it seems probable that the second layer is still represented by the scattered cells of the stratum lacunosum, which if it were continued round the bend formed by the fascia dentata, would come to occupy the region of the granules.

A delicate plexus of primitive beaded fibrils invests these cells, just as was seen to be the case with those of the stratum pyramidalum, and they are closely invested by Golgi-nets.

At irregular intervals on both sides and within this layer are "dark" cells, generally much larger than the granules, sending their long sinuous, delicate branches in all directions among the granules and throughout the molecular layer, likewise the branches of the "dark" cells of the nucleus fascia dentata course among the granules. There is no need to describe them more fully as they are quite similar to those already described.

(9) *Nucleus fasciæ dentatæ.*—Within the hollow of the cap formed by the stratum granulosum are a number of both "dark" and "pale" cells lying scattered about in all directions. They are continuous with and differ only by their irregular disposition from the cells of the stratum pyramidalum, so that no further description of them is necessary.

IV.

Olfactory bulb.—A. Hill, who has made a special study of the olfactory granules by Golgi's method, describes two varieties. The larger has a diameter of 14 μ, the axon appears to run first towards the glomeruli, and then looping on itself runs for a great distance in a central direction. He followed one for 0·7 mm. According to his figure the dendrites of these cells are beset with little lateral projections. In the pictures which I obtain of granules this large variety is, comparatively speaking, rarely seen, the very great majority of the granules belong to his second variety, which are much smaller and, judging from the figure he

gives of them, only imperfectly shown by Golgi's method. The apical branch (the dendrite) is apparently only visible for a very short part of its course, and although the moniliform markings are indicated, yet owing probably to the deposition of silver about the process no idea is obtained of the exceeding delicacy of the fibril connecting the beads. Moreover, I have several times been able to determine that the central process (the axon) after a comparatively short course branches, and branches again in a somewhat dichotomous fashion. The figures of granules reproduced in Barker's book from Kölliker evidently belong to Hill's "type 1," they are shown thickly beset with lateral projections, which the second variety in my preparations never show. It is open to question whether all the small olfactory granules are of the same nature. As with Golgi preparations so with mine, only some stain, but on comparing sections from different cases there is a remarkable uniformity in the number and position of those that stain darkly. They are very regularly sprinkled over the entire granular field.

On the other hand, undoubtedly there are many fewer dark granules in preparations which have only reacted moderately to the stain, and sometimes in the less successful pieces gradational stages can be seen between the granule which only shows a pale blue nucleus, and that which stains out darkly in its entirety. On the whole, I think the balance of evidence is in favour of these small elements all belonging to one class.

The general similarity between the structure of the olfactory lobe and that of the cerebellum is very great, and more minute inspection renders this more apparent. Leaving out of account the peculiar glomeruli which have no representatives in the cerebellum, we get a molecular layer through which the branches of the mitral cells pass, and in which are a number of very small, richly branching "dark" cells, comparable to those seen in the molecular layer of the cerebellum; then a single layer of large cells of the "pale" variety, around the bodies of which the molecular and granular cells form a rich arborisation; next to

this is the granular layer, on superficial inspection strikingly like that in the cerebellum, but which shows several peculiarities, the chief of which seems to be that, in the majority of cases, instead of sending up their axons towards the molecular layer, they send up their apical dendrites. This peculiarity, as well as the presence of the glomeruli, may be associated with the difference between the two organs in their reception of afferent stimuli, the olfactory lobe receiving them from the surface, the cerebellum from the centre.

Hill remarks upon the likeness between the olfactory and cerebellar granules, but states that "many considerations show us that the granules of the bulb cannot be regarded as strictly homologous with the granules of the cortex cerebelli or cortex cerebri. The organ in which they are found is widely dissimilar from the cortex, homologous with a peripheral nervous organ—the retina, and not with a cortical or secondary nerve-field."

It is a striking fact that at all the great sensory higher stations granules are met with in large numbers, and I believe the true granules are met with only at these places. The so-called cells of the cortex cerebri are widely dissimilar in their morphological characters, and I am glad to see that Campbell substitutes for them the term "stellate cells." The olfactory sense has its granules in the olfactory bulb, the visual sense in the retina, and in birds chiefly in the optic lobe. The first of these organs is an early outgrowth from the first vesicle, the optic lobe an outgrowth from the second, and the cerebellum an outgrowth from the third. If there are objections to regarding the olfactory lobe as a cortical or secondary nerve-field, objections which do not seem insuperable to me, it is open to us to regard both it and the cerebellum as a peripheral nervous organ, although the general scheme of construction in olfactory region, cerebellum and cortex cerebri is fundamentally so similar that I would prefer to regard them all three as of central nature.

One more point deserves some consideration before leaving the structure of the olfactory bulb, and that is the

large swellings which characterise the long apical processes of the granules. These swellings are proportionately much larger than those usually seen on the dendrites of the "dark" cells; probably those on the "dark" cells of the thalamus most nearly approach them in size. Fragnito's researches show that not only axons but also dendrites are formed by the longitudinal fusion of different cells, and in embryos these processes still retain more or less evidence of this origin in the form of long oval swellings with the remains of nuclei within them. With my method the swellings on the olfactory granules stain black throughout, so that it is not possible to see any detail within them; nevertheless, I would suggest that they represent the remains of the cell bodies from the fusion of which the process results.

Cornu ammonis.—At the end of the second month in the human embryo, the part which is to develop into the rhinencephalon begins to show itself in the form of an outgrowth from the lower and forward aspect of the fore-brain. It is demarcated at that period from the remainder of the fore-brain by a distinct fissure, which still persists, but in a vestigial form, in the adult brain—the fissura rhinica. This region is specially connected with the sense of smell. Both the regions I am dealing with are parts of this early outgrowth. Although there are some divergencies of opinion as to whether the whole region of the cornu ammonis is concerned with olfactory impressions, it is generally allowed that the gyrus dentatus subserves this function, and the more general opinion inclines to the belief that, if not a secondary, the gyrus hippocampus is at least a tertiary field for olfactory sense impressions. Obersteiner remarks that its degree of development varies with that of the olfactory organ, being quite rudimentary in the dolphin and small in man, whereas in animals of well-developed organs of smell it is large. Hill has shown that, in the Cetaceæ, in which the olfactory bulb is absent, the gyrus dentatus is also wanting. Edinger has shown that phylogenetically the olfactory nerve sends tracts to the superior cerebral centres earlier than any other. In amphibians

which have not otherwise attained to a well-developed cortex, these olfactory connections light upon a well-developed brain cortex corresponding in character and disposition to the cornu ammonis.

We are justified, therefore, in looking upon the cornu ammonis as representing a primitive type of cortex. Its structure shows it to be of a relatively simple type; not only are the cells in closer apposition to one another (a characteristic of all lowly forms of cortex, as Nissl's well-known photographs clearly show), but only three cell layers are represented, the second, the pyramidal (large), and the fusiform. This last, it is to be noticed, is almost completely a "dark" cell layer. J. S. Bolton recognises five layers in the cortex, the first and fourth are fibre layers, the second, third and fifth, cell layers. He makes no attempt to subdivide the pyramidal (his second layer) into small and large cells, the third is his granule (Campbell's stellate) cell layer, and the fifth the polymorphic. This last is the first to develop, the pyramidal the last. His researches go to show that it is the region earliest affected in mental diseases, and he concludes that it subserves the psychic or associational functions of the cerebrum, whilst the fifth probably subserves the lower voluntary functions of the animal economy.

Without entirely accepting his conclusions, in the main they harmonise well with the degree of development of the various layers of the cornu ammonis considered as a primitive cortex. The polymorphic layer (stratum oriens) is extraordinarily well developed, whilst the cells which subserve higher functions are relatively few in number, and in lowly mammals like the rat, mouse and rabbit, closely packed together. In these animals the greater part of the layer which corresponds to the pyramidal of the pallium (the stratum radiatum), containing hardly any cells at all. In man, where the pyramidal cells are less closely packed and more numerous, they begin to encroach on the stratum radiatum, which is relatively narrower than in the lower animals.

Stellate cells, which characterise especially the cortex of the more specialised senses, such as sight and hearing,

are not represented at all, unless, which from their size and configuration seems probable, the second layer of cells and stratum granulosum of the gyrus dentatus are of this variety.

Taking into consideration first its phylogenetic antiquity, second its relative simplicity, and thirdly its structural peculiarities, the cornu ammonis may be taken as, in a sense, the typical form of cortex.

Almost dating from the time when the microscope was first applied to the study of the finer structure of the brain, it was recognised that the different regions of the cortex differed largely from one another in the form and arrangements of their nerve-cells. Since that period a number of observers have made these topographical variations their study, the series culminating in the magnificent work of Campbell, just issued. But in spite of this difference in the form and arrangement of the cortical elements, it will be found on a more minute inspection that the essential structure of all parts is everywhere the same, and the present method, owing to the clearness with which it picks out the finest extracellular fibrils and to its differentiating qualities, renders this uniformity most strikingly evident. In looking over the pictures with which Barker has so generously endowed his book on the nervous system, pictures which in a large number of cases are from Golgi preparations, one is astounded and perplexed at the seemingly enormous complexity in the relationships of nerve-cells to one another. This is largely due, I believe, to the fact that this method, although it picks out details—not of the very finest character, however—with delightful clearness, fails altogether to exert any differential action in its colouring of the cells, so that the branches of both classes appear with the same clearness, crossing and recrossing in the field of vision in bewildering multiplicity.

There are differences, and very important ones, besides tinctorial which distinguish one class of cells from another, and moreover, the differences in staining cannot always be depended upon, but when once the existence of these two great classes has been appreciated, it is comparatively easy to

recognise the other distinguishing characters. The failure of the dendrites of the pyramidal or "pale" cells to stain by my method adds enormously to the clearness and instructiveness of the pictures which it shows. Here, again, as with the cells, besides colour there are other differences in

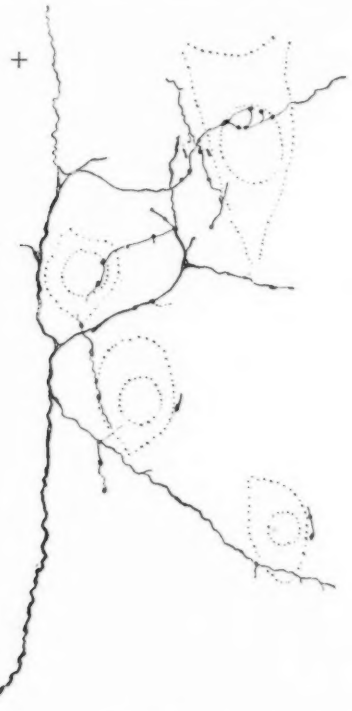

FIG. 11.

Cat's cortex. A stout dark fibre with the characters of a dendrite sending off delicate beaded fibrils to form the pericellular investment over the ganglion cells indicated by dotted lines. At the upper end of the stout black fibre is a fine fibril (+), which has very much the appearance of an axon.

the character of the two classes of dendrites, so that, as when occasionally happens the protoplasmic processes of the pyramids are darkly stained, they can nevertheless be easily distinguished from those of the "dark" cells.

Although I have on more than one occasion given these other distinctive characters appertaining to the two classes of cells, for convenience I will repeat them. The processes of the pyramidal cells—and this is, of course, more striking in the larger ones—pass off gradually from the cell body as comparatively thick branches, which as they proceed from the cell become more and more slender; and further, their tendency is to run in more or less rectilinear or gently

FIG. 12.

Shows pericellular investment of beaded fibrils over two ganglion cells of cat.

curving directions. Above all, they are regular in calibre, showing neither beads or moniliform swellings. The protoplasmic processes of the "dark" cells, on the contrary, spring abruptly either from the cell body or a short stout branch and run a very tortuous course. They start comparatively slender and often run long distances with no or very slight alterations in their calibre. In many cases they are beaded or show little lateral projections.

It is well that sometimes the dendrites of the pyramidal cells stain darkly, for by this means certain points in the relationship of cell processes to one another, not otherwise visible, can be shown with clearness. The delicate little offshoots ending in a knob, which under these circumstances appear all round the dendrite, can in very many cases be traced beyond the apparently terminal knob as a fibril, which is so fine that the intercellular neuro-fibrils, as shown by Cajal's method, are by comparison stout. Usually at a short distance from the dendrite this fibril joins to another, and these, again, to a third, as shown in figs. 8 and 9.

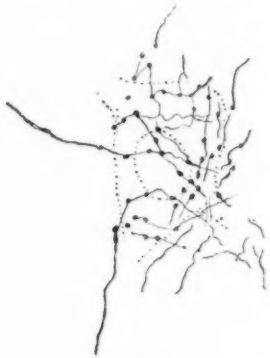

FIG. 13.

Pericellular investment over ganglion cell of cow's cortex, showing greater complexity than is shown in the case of the cat.

So far no appreciable thickening in their calibre has occurred, but after a further course, varying in length, well-marked beads appear and soon the fibre grows manifestly stouter. Such an arrangement represents, I believe, a fibril either passing to the beaded pericellular structure which invests with a greater or lesser complexity every pyramidal cell, or perhaps passing directly into the cell body to swell the number of its intercellular fibrils. In any case, these thorny offshoots from the dendrites of pyramidal cells are essentially different from the very much stouter and sparser lateral projections seen on many of the "dark" cell pro-

cesses, notably those of the striate body. Although these appear generally to be terminal, yet sometimes they also can be traced on into longer or shorter continuing fibres, which, unlike those already referred to, get thinner and thinner as they recede from the dendrite.

One of the reasons why, occasionally, the pyramidal cells stain darkly appears to be due to their Golgi-net coverings, which closely invest the cell and its branchings, and not

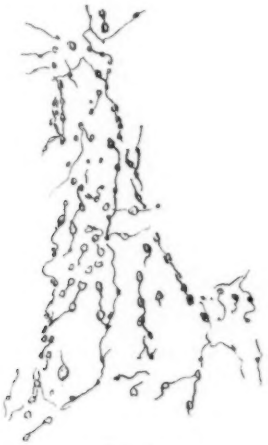

FIG. 14.

Pericellular investment over human Betz cell. Note its complicated character, and the larger size of the beads, which are often hollow or ring-like.

All these figures drawn with the aid of Ziess' camera lucida and magnified about 700 diameters.

only may they take up the stain but apparently hold it also within their narrow meshes.

A great advantage in studying the brain of a small mammal like the rat is that it permits a general survey of the whole organ to be made and, further, the structure is much less complicated than is the case in higher mammals or man. In such a brain stained by my method the fact is very clearly shown, that everywhere the fundamental plan of nervous structure is the same: that is to say, a

combination of cells of two distinct kinds, both as to staining capacity and anatomical characters. One of these classes forms a plexus by means of the branchings of its protoplasmic processes around the cells of the other class. And one of the great points of distinction between the cortex of a lowly mammal like the mouse or rat, and a higher one, as for example the cat, cow or man, is in the disposition of this plexus, which the higher we pass in the scale tends more and more to complexity in its pericellular structure (figs. 12, 13, 14, *C E F.*)

I have now examined a large portion of the human cortex, central nuclei, cerebellum, and stem, the whole surface of the rat's and mouse's brain and their main central region, and various parts of the cat, rabbit and cow, and although the field of observation leaves out many and important parts, the sympathetic system, the spinal cord and posterior ganglia, still the harmonious results in the regions examined tempt me in conclusion to make a few observations on the construction of a scheme of nervous mechanism, which not only fits in with my own observations, but also, so far as I can see, is not incompatible with the observations of other workers on the line of continuity.

The physiological unit of the nervous system is the simple reflex arc apparatus, showing a combination of two or more nerve-cells, with on one side a process in connection with the periphery, and on the other a process in connection with the muscle. An impression or stimulus on being applied to the peripheral, sensory, or afferent process of a nerve-cell, passes centralward along it through the cell body and away by another process, communicating the stimulus to another cell, where, passing peripherally along one of its processes ending in connection with a muscle, it evokes a contraction. This is what we conceive to be the simplest form of nervous mechanism, and so far as our evidence extends it is the only one. The entire principles of the structure and functions of the nervous system are involved in the principles underlying the reflex arc, and if it were possible to understand thoroughly the minute structure and method of combination of the elements concerned in

this arc, we should have a much clearer conception of the physical workings of the entire nervous system. The combination of afferent and efferent nerve-cells and the physical changes accompanying their action is, so far as our evidence permits us to judge, the same in the simplest automatic reflex act as it is in the highest mental one. There would appear to be only one difference between them, not of kind but of complexity; for while the reflex act only involves two or three cells, the mental probably involves hundreds. Unfortunately, at the very threshold of our enquiry we are confronted with difficulties and uncertainties. Leaving aside the differences of opinion concerning the intimate structure of nerve-cells, not only is it a moot point how many cells take a share in the simplest (vertebrate) reflex act, but there is the keenest controversy on the question how these nerve-cells are combined with one another.

In early times when nervous systems first began to be examined by the microscope (Gerlach), the idea was that nerve-cells were continuous with one another through their branches, especially those branches which are termed protoplasmic processes or dendrites. With the introduction of more delicate methods of colouring the finer parts of the nervous system (Golgi's method) the idea that nerve-cells were continuous with one another was gradually abandoned, and until a few years ago the view that almost entirely held the field was that every nerve-cell was anatomically distinct, and that there was no direct union between the branches of any two cells. It was supposed that axons or collaterals broke up ultimately into fine branches, which closely approximated themselves to parts of neighbouring or distant nerve-cells, more especially around the dendrites. The regions where these delicate axonal ramifications came into proximity with dendrites or cell body were termed by Foster synapses, and at these places the nerve impulses were supposed in their passage to jump the spaces between.

All the while that our knowledge of the intimate composition of individual nerve-cells was so defective that for the most part they were regarded as the ultimate units of the nervous mechanism, a view which Golgi's method serves

to encourage, as it usually shows a black silhouette of the cell and its branches, there was no difficulty in drawing up a scheme which could satisfactorily display this supposed relationship of cells to one another, but with the advance of knowledge into the details of nerve-cell structure this became increasingly difficult, so that now on purely *à priori* grounds the idea of the combination of cells by contiguity can be shown to be exceedingly improbable.

It is admitted on all hands that nerve-cells do contain numbers of extremely fine fibrils, and whether these fibrils pass through the cells isolated from one another or connected by still finer lateral threads is immaterial for the present discussion. It can be seen that frequently one of these fine threads leaves a cell, generally by one of its dendrites, and after running as an extracellular fibril for a variable distance it disappears from the section somewhere in the matrix. On the position that I have taken up, that nerve currents of whatever nature they may be are transmitted by means of these threads or neuro-fibrils, it is extremely difficult to form a satisfactory scheme of nerve-cell combinations, according to the theory of contiguity, which will conform to the recognised peculiarities of nerve-cells. If, for example, we endeavour to fill in the fibrillary contents of the cells in the scheme of reflex action as presented in the text-books of the present day, it becomes evident how unsatisfactory it really is. On the afferent side, we must picture multitudes of neuro-fibrils ending blindly either within cell processes or in the matrix; on the efferent, a correspondingly great number beginning in an equally meaningless fashion. Again, the extra-cellular fibrils just referred to, which leave the cells by the dendrites and run free in the surrounding matrix offer considerable difficulties to this idea. Synapses are constituted by the ultimate arborisations of axons or collaterals about dendrites or cell bodies, and never by dendrites about dendrites, or dendrites about axons. Therefore, these stray fibrils cannot be on the way to form synapses about other cells. And if, as is, I believe, universally the case with those who uphold the discontinuous

nature of the nerve elements, the doctrine of dynamic polarisation is insisted on, the difficulties are further increased, for there is no other alternative than to suppose that the fibril springs *de novo* from somewhere in the matrix and passes into the nerve-cell.

Certain features in the development of nerve-cells are opposed to the view that these elements are discontinuous. The axis-cylinder is formed by the conjunction of fusiform cells, and Fragnito has recently brought forward evidence to show that dendrites also are formed in a precisely similar way. Capobianco has shown that even the cell body in the case of posterior spinal ganglia cells is of composite origin. So that a nerve-cell does not correspond to a single cell of any other tissue, but is a compound structure formed by the direct fusion of many.

Of recent years and coincident with newer methods, which reveal structures more delicate than Golgi's, the idea has steadily been gaining ground that there is no discontinuity in the elemental fibrils, but that they run without a break from periphery to muscle. This is an idea which conforms much more readily to the fibrillar structure of nerve-cells, and although it cannot be said to be proved in vertebrates it has been so in the case of those invertebrates which have been made the objects of special study. The newer idea of continuity is, however, very different from the older, the former rests on the continuity of the neuro-fibril, the latter on the continuity of processes which are now known to be composed of a number of these neurofibrils, and my endeavour now is to show that an intelligible scheme of the nervous system can be constructed on this newer basis which harmonises with the anatomical findings of different observers (among whom, as is only to be expected, there is considerable diversity of opinion in regard to many of the details of structures), and which also lends itself readily to some important physiological conceptions, notably, McDougall's fascinating theory of inhibition by drainage.

The existence of a basketwork of nerve-fibres around the Purkinje cells has been known for a long time, and

Ehrlich's vital method shows occasionally a fibrillar investment about the cerebral cells of animals. The method I use, however, not only reveals this structure in the cerebral cells of man, but shows it more fully, and that it is more generally distributed than we should be warranted in inferring from the results obtained by Ehrlich's method. My observations, indeed, show that it is found about all the pyramidal cells of the cortex, the cells of the nuclei of origin of the cranial nerves, and certain cells of the thalamus and corpora quadrigemina, in fact, about all those which fail to stain by my method and which it will be convenient to class together as ganglion cells, in contradistinction to another very widely distributed class which stains deeply, and the branches of which form a plexus whence the fibrillar investment which surrounds the ganglion cells is derived.

At this point it must be mentioned that Bethe also describes a pericellular network structure, the basis of which is the Golgi-net, but this has been shown to be of non-nervous origin. Nevertheless, it may well be that although the Golgi-net itself is a glial structure, it may serve in some way as a support for the passage of delicate fibrils, passing around or into the nerve-cell, as Bethe assumes.

It is evident in any case, that if neuro-fibrils form a pericellular coating to certain cells, they must, if continuous, at some place in their course, come themselves to lie within cells, which in turn are invested with other neuro-fibrils.

In the accompanying diagram (fig. 15) the portion within the bracket represents two ganglion cells with their investing network derived from two intercalary or "dark" cells—*Y*, *Z*. Following neuro-fibril *A* from its origin in the periphery, it passes through an intercalary cell *Y* round a ganglion cell, whence, continuing its course, it sinks into another ganglion cell, passing out by way of the axon to ultimately end in a muscle. And similarly with neuro-fibril *B*. According to the idea of continuity, whether nerve-cells are in connection by means of axonal or dendritic prolongation is of no significance, the foundation of the whole idea is the neuro-fibril, which is met with alike in

both kinds of processes. It will be evident that in the scheme proposed no one ganglion cell can be flung into action without at the same time implicating other cells.

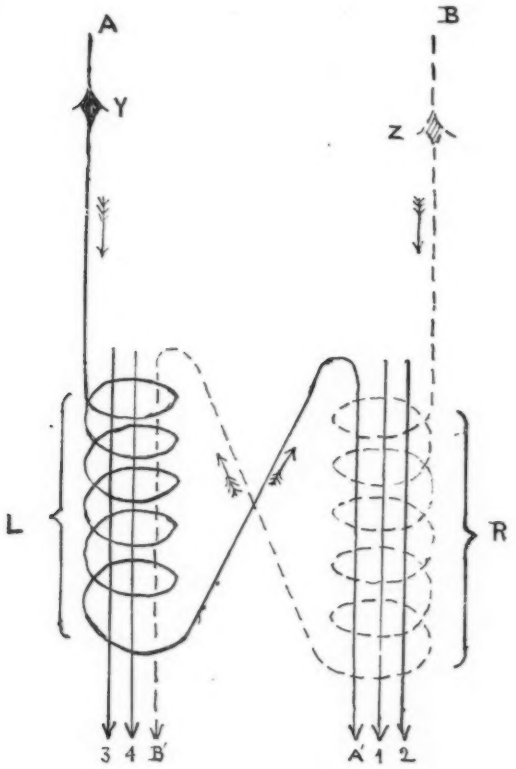

FIG. 15.

Diagram of reflex arc apparatus.

When we come to consider the dynamics of such an apparatus as is suggested, it will be necessary in the first place to grant several assumptions, which, however, are not more speculative than those which have to be assumed in

schemes constructed on the basis of contiguity. In the first place, it must be assumed that the passage of nervous currents passing in the pericellular fibril evokes a current in the intracellular fibrils, so that, for example, a current starting from A not only excites the muscle in connection with A', but sets up currents passing along 3, 4, B'. It is probable that currents are always passing along the fibrils, but it is only when they reach a certain intensity that they are capable of communicating a sufficient stimulus to other fibrils to provoke a visible reaction in the muscle.

The intensity of a current in a fibril may be raised by blocking at the central (muscle) end of a fibril, or by increased stimulus at the periphery; it may be lowered by drawing off the current at the central end, or lessening the irritation at the peripheral organs.

Granting these assumptions, it will be seen how the scheme adapts itself to McDougall's theory of inhibition by drainage. It is obvious that the discharge of the fibres within cell L will lessen the intensity of the stimulus in the encircling fibre of cell R, by drawing off the current from the encircling fibril of this cell, and will in consequence inhibit for the time being discharges from cell R. In fact, if for neurones and supposititious synapses we substitute continuous neuro-fibrils arranged as in the diagram, it will be found that the whole line of reasoning with which McDougall supports his hypothesis is applicable to a nervous mechanism constructed in the manner shown here.

On the basis of this physiological unit, it is possible to construct a scheme of the whole nervous system, which conforms to the newer details revealed by modern special methods and does not conflict with the results of physiological experiment.

The data on which the following scheme is constructed are:—

(1) That neuro-fibrils are continuous, stretching without a break from their origin in peripheral sensory stations to their termination in muscle.

(2) That the most primitive nervous systems consist of cells continuous by their branches and without any distinctive axons (nerve-nets of Bethe).

(3) That the ganglion cells are not true cells. They represent the fusion of many different cells.

(4) That all the ganglion cells are invested by a neuro-fibrillar coat, which necessitates some such arrangement as is shown in (fig. 15).

I would suggest that the true cells of the nervous system are those which by Ehrlich's vital and my method are

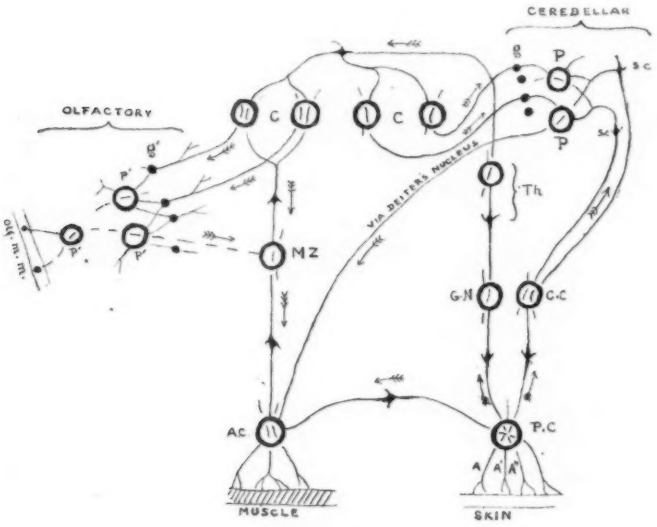

FIG. 16.

Scheme of nervous system, constructed on the idea of the continuity of neuro-fibrils; *PC.*, spinal ganglion cell; *GN.*, gracile nucleus; *CC.*, Clarke's column; *Th*, thalamus; *P*, Purkinje cells; *P'*, mitral cells; *sc.*, small cortical cells of cerebellum; *g*, granules; *g'*, granules of olfactory lobe; *C*, cortical cells; *MZ.*, motor-zone; *AC.*, anterior cornual cell.

darkly stained, those which I have designated "dark" or intercalary and that they represent the development of the cells of the nerve-nets, in which during this process some of the branches have become differentiated to form axons.

In the scheme (fig. 16) the ganglion cells are represented by circles continuous on one side with a fibril from a "dark"

cell, the circle stands for the pericellular fibrillar covering, within are represented intracellular fibrils. In this scheme two cerebellar adjunctive systems are incorporated. The preponderance of "dark" cells in the cerebellar systems and of ganglionic in the cerebral is indicated. A stimulus applied to the skin passes up as nerve waves or currents along the fibrils *A*, *A'*, *A''*. At *PC*, a spinal ganglion cell, it passes around the cell by its pericellular coating, and is drafted off into other channels, becoming more diffused, but reinforcing the nervous currents which pass along by the intracellular fibrils. On the other side of the cell the main currents pass along fibrils, some of which go to cells of the gracile and cuneate nuclei (*GrN*), others to cells of Clarke's column (*CC*) *en route* to the cerebellum.

Following first the current which flows towards the cerebrum, it passes to the thalamus (*Th.*), through the "dark" cells of this region and around the ganglion cells, where it excites nervous currents in the intracellular fibrils which run to the "dark" cells of the cerebrum, and from thence around ganglion cells, along their efferent fibrils (axons) to the motor-zone, the anterior cornual cells and so to the muscles.

Returning now to the spinal ganglia cell *PC.* to follow the current flowing towards the cerebellum, it passes, in a similar way to that before described, through Clarke's column cells, onward to the "dark" cells (small cortical, &c., of the cerebellum), thence both around (basket-works) and within the Purkinje cells to issue along their axons *viâ* Deiter's nucleus, to the anterior cells of the spinal cord. But also from the cerebrum currents are received in the cerebellum which are capable of exciting or modifying the currents in the Purkinje cells, and these currents I suggest pass along fibres to the granules and from thence to the pericellular investments of the Purkinje cells. In a similar way currents are received and issued by the olfactory system—received from the cerebrum through the medium of the granules, and issued from the olfactory lobe to the cerebrum through the axons of the mitral cells.

The above is a brief outline of the main features of the

scheme, no pretence has been made to show all the intermediate stations between the brain and cord, nor the passage of currents from sensory fields of the cerebrum by association tracts to other cerebral fields, nor the afferent fibres issuing from muscles, &c. For the present purpose these are superfluous, and would only render the scheme less clear.

In conclusion, one point remains to be briefly touched on, and that has reference to the degeneration which occurs in a nerve-sheath after its severance from a ganglion cell. There are grounds which afford some justification for regarding the chromatoplasm of these cells as reserve sources of nourishment and energy for the nerve-fibres. On these grounds, the scheme submitted provides that degeneration occurring after section of nerve-trunks would occupy the site it should do as shown by the result of experiment.

Although the fact ascertained by Gotch and Horsley that electrical variations will pass from the dorsal to the anterior root, but will not go in the reverse direction, may favour the idea of discontinuity in neuro-fibrils, yet it does not necessarily imply discontinuity, and I believe could be reconciled to a scheme of nervous structure on the lines of continuity, such as I have here suggested. But in any case the question whether neuro-fibrils are continuous or discontinuous is not one that can be definitely settled by experiment. The ultimate appeal must, I submit, be to microscopical anatomy.

Addendum.

From a comparative study of different regions of the human cortex by the present method I am not able to agree with A. W. Campbell in the subordinate position to which he assigns the frontal (his præfrontal) lobe. The ganglion cells are here sparser and more widely separated from each other, it is true, but reasoning from analogy this does not appear to be an indication of stunted development, although it would coincide with incomplete development.

Comparing, as Nissl has done, a series of cortices, beginning with the lowliest mammals and passing to the highest, it is found that, as the scale ascends, the cells come to lie wider and wider apart. Thus this fact which Campbell uses to support his contention seems to me to bear quite a different interpretation. Surely that part of the cortex which subserves the highest intellectual functions will be that latest evolved, and therefore in the least organised condition, and if so the comparative paucity of ganglion cells really favours the view of those who, like Ferrier and Bianchi, assign the highest mental functions to this part of the cortex. Campbell, although he refers also to a paucity of fibres in this (the præfrontal) region, is only referring, I take it, to those displayed by the non-specific stains he uses. Examinations of sections of this region treated by my method show here a wealth of intercellular fibrils probably greater than in any other region of the cortex. Under a low power the gray matter appears to be thickly sprinkled over with tiny specks which, with higher magnification ($\frac{1}{12}$ oil immersion), are seen to lie in the course of neurofibrils, more delicate, perhaps, than are to be found in any other part. A comparison between this plexus in the præfrontal region and the intercellular plexus of the calcarine cortex shows very strikingly that in this latter region not only are the fibrils much less numerous, but they are stouter, or, at least, appear to be so, owing to the comparatively large beads which lie in their course. Such facts as these lend colour to the view which Nissl holds as to the importance of the development of the gray matter for mental capacity.

A comparison of the intimate structure of the gray matter in man and lower mammals, reveals differences which still further support Nissl's view, whilst in the rat, for example, in those parts (cornu ammonis and pyriform lobe) where the intercellular plexus is most developed, not only is this plexus relatively less rich in fibrils, even when compared with those regions of the human cortex where it is probably least developed, but its individual fibres are comparatively much coarser than are to be found in man.

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Microphotographs.

Plate I.

(*A*) *Olfactory bulb* (rat), + 400.—Upper part of picture shows the molecular layer with the dark moniliform processes of granule cells passing across it. In the centre of figure is the mitral cell layer—large, colourless cells with dark nuclei—delicate beaded fibrils are seen around the body of the mitral cell in the centre of the picture. The lower half of the picture is occupied by granules which are arranged in rows. The greater number of the granules have a colourless cytoplasm and pale nucleus, but one here and there is stained darkly showing long moniliform apical processes.

(*B*) *Stratum oriens* (rat), + 300.—Passing obliquely across the upper part of the figure on the left side, longitudinal fibres of the alveus are dimly indicated, among which beaded protoplasmic processes from the cells of the stratum oriens are mingled, these latter striking in at right angles to the course of the alveus fibres. The great majority of the cells of the stratum oriens are "dark" cells. They show no regular arrangement, and their delicate moniliform processes pass abruptly from the cell body and form a loose-meshed plexus throughout this layer.

Plate II.

(*C*) *Stratum pyramidalum* (rat), + 400.—Section passes through the body of the cells at right angles to their long direction. The pyramidal cells are shown as somewhat pentagonal clear spaces, closely packed together, and with a slightly stained nucleus. The smaller white spaces in darker background are apices of pyramidal cells cut across. Five or six "dark" cells are seen scattered about among the pyramids, from whence proceed the dark moniliform branches which wind about among the pyramids.

(*D*) *Stratum pyramidalum* (rabbit), + 600.—Section passes through bodies of the pyramidal cells, which are closely packed together, and surrounded by a plexus of primitive beaded fibrils.

PLATE III.

(*E*) *Frontal cortex* (rat), + 300.—Pyramidal cells appear as colourless definitely orientated bodies, with moderately dark nuclei. "Dark" cells with no definite orientation and thin sinuous branches winding about over the field. The long cell about the centre of the picture shows an ascending axon.

(*F*) *Thalamic region* (rat), + 400.—"Pale" cells with moderately dark nuclei scattered irregularly (no definite orientation). "Dark" cells much larger than the "pale," giving off branches forming a plexus about the "pale" cells.

Figures *E*, *F*, should be compared with fig. *C*, showing similar plan of structure in all three.

THE ABDOMINAL REFLEX IN ENTERIC FEVER.

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THE value of the abdominal reflex in clinical medicine was first pointed out in 1876 by Rosenbach[1] [14], and has since been emphasised by numerous neurologists, especially by Strümpell [17], Babinski [1], and van Gehuchten [18], more particularly in organic hemiplegia and paraplegia, disseminated sclerosis and tabes.

This reflex, however, has not received such general recognition in the medical world as has been given to the tendon reflexes, and until quite recently its investigation had been confined to nervous diseases. Some authorities, indeed, are inclined to assign little value to its presence or absence. Thus Oppenheim [12] says that it is inconstant in health in some individuals, and is therefore not of much practical importance. The falsity of this statement was proved by Müller and Seidelmann [11], who explored the reflex in 3,000 persons who were free from nervous or abdominal disease. With one exception, in which only a single and that not a complete examination was made, the reflex was present in all. The writers concluded that when the abdominal reflex is absent in a young patient of either sex it is most probable, if the proper technique has been employed, that there is a pathological cause, which may be either disease of the nervous system or acute infection of the abdominal organs. Among the first to have noted affection of the reflex in acute abdominal disease was Jamin [7], who drew attention to its loss or impairment on the right side of the abdomen in perityphlitis, gall-stone colic and cholecystitis. In chronic abdominal

[1] The absence of the abdominal reflex is sometimes known as Rosenbach's sign. Babinski, Soc. de Neurol., January 7, 1904.

disease such as ascites, tuberculous peritonitis, and tumours, active reflexes, he declared, could often be obtained. He quotes Bodon [4] as finding the same in diseases of the female pelvis, the reflex being much more frequently affected in acute disease in that region than in chronic affections. The only systematic observations hitherto published on the condition of the abdominal reflex in enteric fever were carried out by Sicard [16] on twenty-six children. In twenty-two the reflex was modified, being absent on both sides or notably diminished and rapidly exhausted on admission to hospital, and remaining such during the whole of the febrile period. Unilateral absence of the reflex, most often on the right side he found was not exceptional during a certain period of the disease. In two cases the modifications were very transient, and in the remaining two the reflex was not affected at all.

The abdominal reflex has by some neurologists been subdivided into three parts: an upper segment called the epigastric, an intermediate segment, the abdominal, and a lower segment, the hypogastric. Sicard proposes the terms epigastric, para-umbilical, and hypogastric zones. For practical purposes it will be well to follow Müller and Seidelmann in adopting Oppenheim's division of the reflex into a supra-umbilical and infra-umbilical zone. In enteric fever, it is the infra-umbilical reflex that is the first to disappear and the last to return. The supra-umbilical response may remain active throughout the disease, or, as more commonly happens, a slight response, rapidly exhausted after a few stimuli may be obtained in the epigastric region or sides of the abdomen alone, when stimulation of the rest of the abdomen provokes no response.

The present paper is based on observations made on sixty patients, all of whom were certified on admission to hospital to be suffering from enteric fever. During the acute stage, the observations were made daily. Their ages and sexes are given in the annexed table:—

TABLE I.

M. F. (1-10)	M. F. (11-20)	M. F. (21-30)	M. F. (31-40)	M. F. (41-50)	M. F. (51-60)	M. F. (61-70)
6 6	8 5	7 7	8 2	4 4	1 1	1
12	13	14	10	8	2	1

In forty-five cases the diagnosis of enteric fever was confirmed; the remaining fifteen patients were found to be suffering from other diseases, viz., lobar pneumonia, two cases, infective endocarditis, two cases, influenza, two cases, leucocythæmia, cirrhosis of liver, cholelithiasis, appendicitis, carcinoma of the peritoneum, tuberculous peritonitis, erythema nodosum, pleurisy, bronchitis, one case of each. In only three of these fifteen cases was the abdominal reflex affected.

In the first case, which was one of suppurative appendicitis, the abdominal reflex was present and fairly active except in the right iliac region, where it was completely absent. Within a week, however, of the evacuation of the abscess, the reflexes on both sides of the abdomen were equal and active. In the second case, where the patient died on the day following admission, acute cancerous peritonitis was found *post mortem* to have supervened upon carcinoma of the stomach. No abdominal reflex was present in this case. The third case was that of a woman, aged 54, suffering from lobar pneumonia. Neither during the febrile period, nor in convalescence, could any abdominal reflex be elicited, but owing to her age and the wrinkled condition of her abdominal wall, which was the result of twelve pregnancies, the absence of reflex was not regarded as due to her disease. That the abdominal reflex is not affected in chronic abdominal diseases is illustrated by the fact that it was present in the cases of tuberculous peritonitis and cirrhosis of the liver.

In the forty-five cases of enteric fever the abdominal reflex was affected to a varying degree in forty-two (93·3 per cent.); it was completely lost in thirty-one (68·8 per cent.); impaired in a greater or less degree short of absolute extinction in eleven cases (22·2 per cent.); unaffected in three cases only. These figures testify that affection of the abdominal reflex in enteric fever is a more frequent phenomenon than the loss of the tendon reflexes in diseases associated with peripheral neuritis, and that it therefore has a better claim to rank in the semeiology of enteric fever than impairment of the knee- and ankle-jerks has in that of diph-

theria.[1] On the other hand impairment of the abdominal reflex in enteric fever is of much shorter duration as a rule than that of the tendon reflexes in diphtheria, which once lost often take many weeks or months to return.

In the great majority of cases the abdominal reflex in enteric fever resumes its normal activity as convalescence becomes established.

TABLE II.

Showing relation of abdominal reflex when first examined to stage of disease.

	Reflex active.	Reflex impaired.	No reflex.
1st week of disease	1	4	2
2nd ,, ,, ...	3	6	6
3rd ,, ,, ...	1	8	7
4th ,, ,, ...	1	1	3
5th ,, ,, ...	1	0	1

Of the twenty-six patients in whom the abdominal reflex was either active or sluggish only on admission, thirteen lost the reflex completely for varying periods of time. In only three cases was the reflex active throughout. In two of these cases, a boy and girl, aged 11 and 7 years respectively, it was first examined five days after the evening temperature had become normal; in the third, a boy of 6, where the disease assumed a mild form, the reflex was first examined on the seventeenth day, thirteen days before the evening temperature became normal. As will be seen from Table II., in the majority of cases the abdominal reflex was affected in a varying degree when the patient was first admitted to hospital. In almost every case where recovery occurred, an active response was persistently present before the patient was discharged. In a small number of cases, but only in those first seen in the early stage of the disease, the abdominal reflex was very brisk, thus resembling the condition of the knee- and ankle-jerks sometimes met with in cases of peripheral neuritis, *e.g.*, in diphtheria.

[1] In 100 cases of diphtheria the knee and ankle jerks were affected in sixty-nine and forty-seven cases respectively, *vide* author's paper in BRAIN, 1905, p. 68.

In this connection it may be remarked that it is only in the severest cases of diphtheritic paralysis that the superficial reflexes are lost, *e.g.*, those in which the pharynx, larynx, and diaphragm are involved. Such loss in cases that recover is of short duration, the superficial reflexes returning long before the re-establishment of the knee- and ankle-jerks to their normal condition.

TABLE III.

Showing relation of abdominal reflex to pyrexia.

	Cases.
Return of abdominal reflex to normal activity and establishment of normal evening temperature in same week ...	19
Evening temperature normal three weeks before abdominal reflex became active	3
Evening temperature normal two weeks before abdominal reflex became active	5
Evening temperature normal one week before abdominal reflex became active	5
Evening temperature normal one week after abdominal reflex became active	2
Evening temperature normal two weeks after abdominal reflex became active	1
Reflex never active	2
Reflex not affected	3
Deaths	5

Before the abdominal reflex resumed its normal activity, there was often a transition period in which it might be fairly active one day and not be present the next, or be limited to one area, usually the epigastric or lateral abdominal aspect. That the abdominal reflex is not affected by maintained pyrexia apart from acute abdominal or nervous disease I have proved by finding it active, not only in the fifteen cases mentioned, but by examination of the variety of febrile conditions that are to be found in a large fever hospital, *e.g.*, scarlatina, measles, diphtheria, varicella, and their febrile complications, serum pyrexia, &c.

The time during which the abdominal reflex was completely absent varied from one day to twenty-one days. The

average time in thirty-one cases was 6·5 days. The time that elapsed before the reflex regained its normal activity was of course much longer, and is shown in the following table :—

TABLE IV.

Date at which the abdominal reflex became active.

	Cases.	Average age of patients.
1st week	—	—
2nd ,, ...	—	—
3rd ,, ...	5	10·2
4th ,, ...	3	24·6
5th ,, ...	9	21·8
6th ,, ...	5	30·8
7th ,, ...	6	30·3
8th ,, ...	3	38
9th ,, ...	1	51
10th ,, ...	2	22*
11th ,, ...	1	12†

* Very severe attacks.
† Two relapses occurred in this case.

From this table have been excluded five fatal cases, three cases in which the abdominal reflex was not affected, and two cases in which the reflex remained sluggish till the time of the patient's discharge. The practical value of the abdominal reflex lies not only in its diagnostic importance, but also in its being a guide as to the state of the disease.

Re-appearance of a lost reflex or increased activity of a sluggish response was often found to coincide with a change in the character of the stools, the pea-soupy discharges being replaced by semi-formed and finally formed fæcal matter as the response became more active.

Persistent absence of the reflex in spite of lysis in a patient under 50 should, in the absence of disease of the nervous system, suggest that a relapse was imminent, and should contraindicate increase in the diet.

The abdominal reflex thus furnishes another clinical method for prognosis in enteric fever, and may suitably be studied in conjunction with the characteristic changes in the urine. I have often found that re-appearance of a lost

reflex or increased activity of a sluggish one corresponded with a disappearance of the diazo-reaction, and still more closely with that of Russo's methylene blue reaction from the urine. Such disappearance, as I have shown elsewhere, shortly precedes or coincides with the commencement of lysis.

The condition of the abdominal reflex is of especial interest in cases of recrudescence of pyrexia in convalescence from enteric fever. In eight cases out of the forty-five, six were proved to be definite relapses not only by the re-appearance of one or more of the classical triad of symptoms, viz., the steplike temperature, enlargement of the spleen, and rose spots, but also by marked change in the condition of the abdominal reflex. In all the six cases the reflex which had previously been active was impaired; in four it was completely lost, in two it became limited to the supra-umbilical region and difficult to elicit. In the remaining two cases, wherein the pyrexia was not due to a relapse of enteric fever, but to erythema nodosum and pulmonary tuberculosis respectively, the abdominal reflex preserved unimpaired the briskness of convalescence.

A striking concomitant symptom is a modification of the normal ticklishness of the individual, which, though most marked in women and children, is not confined to them. In the earliest stage of the disease, and in conjunction with an exaggeration of the abdominal reflex, the normal ticklishness may be increased, but it soon becomes blunted, and prolonged stimulation of the skin provokes no protest. This change is due to the general dulling of the sensorium by the typhoid toxins which occurs to a greater or less degree in almost every case of enteric fever.

As convalescence becomes established and the reflex returns, the normal ticklishness re-appears also, so that it may be impossible to judge of the condition of the reflex owing to the spasmodic contraction of the muscles before the stimulus is directly applied to the surface of the abdomen.

Geigel [6], in the investigation of cutaneous reflexes in healthy subjects, met with similar results. In sensitive persons, he says, the mere approach of the percussion

hammer to the skin is often enough to elicit a well-marked reflex.

TABLE V.

Relation of the abdominal reflex to knee- and ankle-jerks.

	Knee- and ankle-jerks normal or exaggerated.	Knee- and ankle-jerks sluggish.	Knee- and ankle-jerks lost.
Abdominal reflex normal ...	2	1	—
,, sluggish...	9	1	1
,, lost ...	9	19	3
	20	21	4

In these cases there was practically no dissociation between the activity of the knee- and ankle-jerks, which are, therefore, considered together.

Table V. shows that, in the majority of cases in which the abdominal reflex was affected, the deep reflexes were also impaired. In a minority of cases the deep reflexes remained unaffected, or even exaggerated throughout the disease.

There is not, therefore, in enteric fever the striking antagonism between the condition of the superficial and deep reflexes to which Rosenbach and van Gehuchten have drawn attention, that obtains in organic diseases of the nervous system, and notably in hemiplegia, spastic paraplegia, and disseminated sclerosis.

In fifteen cases ankle clonus was present.[1] During the presence of clonus, the state of the abdominal reflex was as follows: in three it remained active, in twelve it was affected, being completely lost in six, and rendered sluggish in six. In eleven of the fifteen cases there was the normal flexor response; and in four Babinski's sign was obtained, but was present only for a few days, being soon replaced by a flexor response.

The only other mention which I have been able to find in literature of Babinski's sign in enteric fever is given by

[1] Definite ankle clonus, with ill-marked or entirely absent knee-jerks and ankle-jerks, occurred in five of my cases. This dissociation of the tendon reflexes and ankle clonus was noted by Maurice de Fleury [5], in 1884, as occurring in typhoid, and more recently by Beaujeu [2].

Kornilow [8], who cites a case in which it was present without any other symptom indicating disease of the pyramidal system.[1]

Relation to sex.—Until repeated pregnancies have produced their usual effect upon the abdominal wall the reflectivity of the female is usually more acute than that of the male.

Below the age of fifty, however, in spite of a fat and wrinkled abdomen, an active reflex could, in convalescence, be readily obtained in nearly all the cases.

Relation to age.—Undoubtedly of more importance than the question of sex is that of age as regards the activity of the abdominal reflex. In early infancy the abdominal reflex in the normal child is ill developed, but enteric fever is fortunately rare. After that period the abdominal reflex in health is always active, though its presence may be masked by crying, which an attempt to elicit it may produce.

Various circumstances account for the briskness of the reflex in childhood.

(1) The inhibitory power of the cerebrum is less marked in the child than in the adult. Sharkey [15] has drawn special attention to the activity of the reflexes in the young, and quotes Virchow's statement in explanation, "The child leads a purely spinal cord existence."

(2) Certain factors, as Bertolotti [3] has pointed out, are absent in children which in some adults may interfere with the production of the reflex, *e.g.*, obesity and laxity of the abdominal walls, such as occurs in multiparæ.

Under these circumstances it is natural that the abdominal reflex should be less frequently completely abolished in the child than in the adult, and that, in cases where it is lost, it should re-appear at an earlier period than when so affected in older persons. Thus, in eleven patients under 15, the reflex was lost in seven cases, or 63·6 per cent.; in thirty-four patients over 15 the reflex was lost in twenty-four cases, or 70·5 per cent.

[1] Its occasional presence in scarlatina, in which, as in enteric, it indicates a temporary perturbation of the pyramidal system by the specific toxins, has recently been pointed out by Kiroff, *Rev. Neurol.*, 1905, p. 1,119.

In the seven cases under 15 the average duration of the complete loss of the reflex was 4·5 days, while in the twenty-four cases over 15 the average period was 7·1 days.

From Table IV. it will be seen that, as a rule, the re-establishment of the reflex to its normal condition took place sooner in younger than in older persons. In healthy adult life, though no longer possessing the briskness of childhood, the abdominal reflex usually remains active till the age of 50, after which period it tends to disappear, thus resembling the tendo-Achillis jerk. Enteric fever, however, after 50, is almost as uncommon as in infancy, so that the question as to whether the loss of an abdominal reflex is normal or pathological will not often arise.[1]

In striking contrast to Sicard's cases, the great majority of my patients were adults (*v.* Table I.), but, with the two exceptions already mentioned, all gave an active reflex before discharge from hospital. Of the two cases one was a woman, aged 25, in whom repeated pregnancies had produced a toneless condition of the abdominal wall, while the other was a man, aged 45, in whom the panniculus adiposus was abnormally well developed.

Apart from local changes in the abdominal wall that tend to interfere with the activity of the reflex, it is reasonable to suppose that there may be a premature loss of the normal reflex apart from definite organic disease, just as there are premature changes in the arteries, the hair, and the teeth. On the other hand, in others the abdominal reflex may even in spite of unfavourable local conditions be retained beyond the usual date for its disappearance. Thus, a woman, aged 51, in spite of a very fat abdomen, furrowed by lineæ gravidarum, presented in convalescence an active reflex associated with well-marked ticklishness.

The pathogeny of the affection of the abdominal reflex is somewhat obscure. It is explained by Jamin [7] as a spastic reflex paralysis; Müller and Seidelmann [11] hold the same view. Sicard [16] attributed the affection to sympathetic

[1] During the last six years, out of an annual average of 1254·8 enteric patients admitted to the Metropolitan Asylums Board's Hospitals, the percentage of cases over 50 has not exceeded 1·7.

irritation at the level of the intestinal ulceration affecting the gray matter and producing peripheral disturbance of the abdominal muscular tone. He further points out that the abolition of the reflex might either be due to a pathological muscular contraction, or, on the other hand, might coincide with a flaccid condition of the abdominal musculature.

The developmental connection of the activity of the abdominal reflex with the pyramidal tract,[1] the frequent association of impairment of the reflex with ankle clonus, and more rarely with Babinski's sign, seem to indicate a perturbation however transient of the pyramidal system. Whether the spinal motor centres are affected directly by the typhoidal toxins as suggested by Massalongo [9] to account for the exaggeration of the deep reflexes in enteric fever, or by reflex irritation as maintained by Jamin, is a matter not easy to decide. In the examination of the abdominal reflex the following practical points must be borne in mind :—

(1) The abdominal muscles must be relaxed. Hence it is impossible in some cases to judge of the condition of the reflex. The patient may be crying or in fear of being tickled contract the abdominal muscles.

(2) A single negative result is of little value. Various circumstances, among which is probably on some occasions a faulty technique, may prevent the reflex being elicited, though its presence is potential. For the proper investigation of the abdominal reflex it is essential that frequent, best daily, examination should be made throughout the course of the disease. It is from neglect of this precaution that in former days inaccurate conclusions as to the condition of the reflex in enteric fever were made. Angel Money [10], for instance, maintained that an exaggeration of all the reflexes including the abdominal was met with in typhoid fever. A similar statement was made by Strümpell [17].

The best method I have found is lightly and rapidly to stroke the skin of the abdomen with the end of a penholder.

[1] Bertolotti [3] has shown that in infants the abdominal reflex was almost always absent where there was an extensor plantar reflex, while in such cases a dorso-lumbar reflex was frequent. He then enquires whether cutaneous reflexes of the extensor types in children whose pyramidal tract is in a state of incomplete myelinisation are more prevalent than the flexor type.

Ticklish and apprehensive patients should be engaged in conversation or their attention otherwise diverted. Jendrassik's method of reinforcement so suited for the eliciting of sluggish tendon jerks is inapplicable here, since, as Müller and Seidelmann have shown, with the clasping of the hands is associated a simultaneous contraction of the abdominal wall.

The examination is easy to carry out, causes no distress to the patient, and from its considerable diagnostic and prognostic significance deserves to form part of the routine examination of every case of enteric fever.

Summary.

(1) The abdominal reflex is affected in a very large number of cases of enteric fever, the percentage of cases in which it is entirely lost exceeding those in which its normal activity is diminished only.

(2) From its absence under 50 being confined to certain nervous diseases and acute abdominal conditions, notably appendicitis and enteric fever, the absence of the abdominal reflex in a given case of continued pyrexia in any patient below 50 is of considerable diagnostic value.

(3) The comparatively transient nature of the affection of the abdominal reflex in enteric fever is a striking contrast to the more chronic affection of the knee- and ankle-jerks in diseases associated with peripheral neuritis, *e.g.*, diphtheria.

(4) Return of a lost reflex, and *a fortiori* resumption of its normal activity, are a valuable indication of commencing convalescence, and often correspond with lysis and characteristic changes in the fæces and urine.

(5) The objective sign of return of the reflex is often associated with the return of the subjective feeling of ticklishness normal to the individual.

(6) In re-appearance of pyrexia in convalescence, the condition of the abdominal reflex is a valuable index of the nature of the pyrexia.

(7) No constant relation exists between the condition of the abdominal reflex and that of the tendon reflexes.

(8) The frequency, degree, and duration of impairment of the abdominal reflex are, as a rule, in direct proportion to the age of the patient.

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A FAMILY IN WHICH SOME OF THE SIGNS OF FRIEDREICH'S ATAXY APPEARED DISCRETELY.

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In 1861, Friedreich [10] published the records of the disease to which his name has been applied. It is a disease characterised by the following signs and symptoms: Slow and progressive ataxy of the four limbs, usually attacking several children of the same family—dating often from a very early age—commencing in the legs, extending gradually to the trunk and arms, the muscles of the larynx, those of the tongue and of the eyes; weakness of the legs, increasingly difficult gait, static ataxy (Friedreich); difficulty of articulation, nystagmus, spinal curvature, paralytic clubfoot, abolition of knee reflexes, no sensory disturbances, absence of oculo-pupillary anomalies, and of lightning pains, integrity of sphincters.

The pathological process is a sclerosis of the posterior and lateral columns of the cord, together with changes in the vesicular column of Clarke. The sclerosis extends throughout the length of the cord upwards to the bulb, implicating the hypoglossal nuclei. It is most marked in the posterior columns which are involved from the lumbar region upwards, less marked in the crossed pyramidal and direct cerebellar tracts, and the posterior roots usually escape. A further point which may be of importance, and one which has frequently been noted, is the extreme smallness of the spinal cord and cerebellum.

Such is a brief outline of the symptomatology and morbid anatomy of Friedreich's ataxy. The chief point to bear in mind about its etiology is the tendency it shows to run in

families; usually several brothers and sisters are affected. Direct transmission from parents to children is rare, owing to the early age at which patients are attacked, generally about the age of fourteen. Occasionally, however, it can be traced back, as in Rutimeyer's case [35], to the great-great-great grandfather. It is not a very rare disease, for within thirty years of the publication of Friedreich's first cases upwards of two hundred others had been recorded. But an examination of some of these shows that observers had very loose ideas as to the true meaning of Friedreich's ataxy, for there are many cases recorded which bear but very slight resemblance to the clinical signs laid down by Friedreich as typical of the disease he described.

Ladame [19] in a paper published in 1890, reviewing the literature of his day, upheld the existence of Friedreich's ataxy as a definite clinical entity, and protested against the inclusion of any case which did not conform to the classical type described by Friedreich, and he even refused to admit into the group any case in which the knee-jerk was not abolished. In doing this he undoubtedly called attention to the extremely definite set of symptoms which characterise Friedreich's ataxy, but he left all those cases which he excluded from his diagnosis in a most unsatisfactory position. It is recognised now that many of these cases show distinct relations to Friedreich's ataxy, while even those that differ from that condition often seem to be linked to it by intermediate forms; it seems as if some families inherit a factor which predisposes individual members to a degeneration of certain parts of the nervous system, and that the localisation of these degenerative processes determines the clinical signs of each particular case. It is thus quite possible that the ataxy described by Friedreich, and many of the other forms of family nervous disease, are not distinct diseases at all, but merely clinical expressions as it were of changes, affecting different parts of the central nervous system, induced by the afore-mentioned hereditary factor.

In typical cases of Friedreich's ataxy the knee-jerk is always abolished, but there are some instances recorded which, although the knee-jerk is retained, resemble Fried-

reich's ataxy in other respects. As a rule these cases occur in families in which the other members conform to the usual type. The explanation is usually given that the retention of the knee-jerks depends upon the distribution of the sclerosis in the posterior columns, and that they are retained in those cases where that portion of the cord concerned in their production has not been invaded by the degenerative processes.

Tressider [43] records a family in which two sisters, aged 50 and 47, and a brother, aged 42, were affected with an ataxy at the age of 12, 13, and 19 years respectively. They were clinically all cases of Friedreich's disease, though the knee-jerks, which were absent in the elder sister, were retained in the younger sister and in the brother.

Brock [6] records a similar instance in a family under his care. The family tree showed a tendency to the development of nervous disease in several branches, but he was only able to examine three individuals—two brothers, aged 40 and 27, and a nephew of 15. They were all undoubted cases of Friedreich's disease, but the second brother retained his knee-jerks. It is noteworthy that in the case of the nephew who was an early, but definite, example of the disease, the knee-jerks were lost.

Other and similar cases have been observed by Senator [37] and Hodge [17]. In Senator's case, the knee-jerk, though present at an early stage of the disease, disappeared later [36]. Small [39] records the cases of four sisters, aged 26, 25, 24 and 22 years, where the onset of ataxy was noted at 13. Knee-jerks were present in the third case, absent or doubtful in the first, absent in the others. The first three cases showed the presence of some optic atrophy which is not a sign that is usually observed in ordinary Friedreich's ataxy, and its presence here seems to link these cases to some shortly to be mentioned where it frequently occurs.

Sometimes the knee-jerks can be obtained in all the affected members of a family. Byrom Bramwell [4] mentions two brothers he had an opportunity of examining in which this was the case. At the time of examination

they were 25 and 19 years old, and the disease had begun at the age of 13.

Sometimes the stress of the disease seems to fall on the lateral columns especially, so that the knee-jerks are exaggerated and ankle clonus can be obtained.

Gladstone [13] saw two brothers, one a typical example of Friedreich's disease, the other similar in all respects, but possessing exaggerated knee-jerks and ankle clonus.

Hodge [16] similarly reports a family in which two sisters and a brother, 44, 40, and 30 years old respectively, were examples of what appeared to be ordinary Friedreich's ataxy, but in all the cases the knee-jerks were exaggerated, and ankle clonus was obtained. Besides these signs, pointing to implication of the lateral columns, they showed marked pes cavus, and two of them had wasting of the small muscles of the hands and a condition of main en griffe.

Although the lateral columns may be affected to such an extent that the legs become rigid, yet the knee-jerk is not always exaggerated or even present.

Smith [40] reports at length a family in which five members were affected with Friedreich's disease, and one girl, in whom the disease had lasted thirteen years, and who was 26 at the time of examination, had spasticity of the legs, but no knee-jerks. This family will be referred to again, but, in passing, it may be mentioned that the father, in transmitting the disease to his children, did not apparently entirely escape, but, at the same time, it must be admitted that the disease he suffered from bore little resemblance to that affecting his daughters. At 66 he became completely ataxic, and was hardly able to walk; he had exaggerated knee-jerks and vertigo. His arms were not affected, his electrical reactions were normal, sensibility was unimpaired, and he had a girdle sensation. There was no history of syphilis.

Nolan [30] records three very interesting cases. They were the third, fifth, and eighth children of a family of eight. All the others (except one dead of scarlet fever) were alive and healthy. But these three were congenital

imbeciles, and "undoubted examples of Friedreich's ataxy." The eldest and youngest conformed to the usual type, but the other had exaggerated knee-jerks and ankle clonus. Two of them also showed wasting of the small muscles of the hand. It is surely something more than mere coincidence that these three children in an otherwise healthy family should be examples of Friedreich's disease, and also, at the same time, show signs of mental deficiency; and one cannot help thinking of the idea that has been put forward, that these family nervous diseases are due to degenerative processes taking place in a nervous system that is not fully developed.

The cases that must now be considered differ from the foregoing in that there is no trace of any involvement of the posterior columns; they are examples of a family spastic paraplegia.

Now, spastic paraplegia is not a very uncommon condition in young children as a sequela to destruction of cells in the cerebral cortex, and in such cases there is frequently a history of convulsive seizures. I have thought it advisable, therefore, to exclude all those cases in which convulsions are mentioned as having preceded the onset of the disease.

Bayley [1] was able to examine nine cases of a spastic paraplegia occurring throughout five generations of one family. Five other cases were known to exist in this family, and it was stated that more still were to be found in distant branches. Of the nine that Bayley saw, one had been affected since birth, six were affected between the ages of 3 and 7, and two at 23 and 26. The disease began with a gradually-progressing paraplegia with rigidity. There was no atrophy of muscles, no static ataxy, no alteration of sensation, no interference with the function of the bladder or rectum. The arms were quite unaffected, the pupils were equal and active, and there was no diplopia. There was marked talipes equinus, necessitating tenotomy in one case, and talipes equino-varus in two others. In the case that began at 26, and which had lasted four years at the time of examination, the knee-jerks were absent, but in all the others they were present, and ankle clonus was the rule.

Tooth [42] described two families which resembled Bayley's in the principal features. In the first family, two brothers, 29 and 24 years old, developed stiffness of the legs at the age of 15. The younger showed at the time of examination great rigidity of the lower limbs without loss of muscular power, very exaggerated knee-jerks, but ankle clonus was not obtained on account of the rigidity. The gait was spastic in type, and Romberg's sign was absent. The arms showed no rigidity, wasting, inco-ordination or tremor. Tactile sensibility was perfect, and there was no girdle sensation. The pupils reacted to light and accommodation, there was no nystagmus, and the fundi were normal. Speech became affected with a stammer after the onset of spasticity. There was slight pes cavus, and the sphincters were intact.

The elder brother showed a condition similar, but more advanced; walking, in his case, had become impossible, and the knee-jerks were so exaggerated that each tap produced a thigh clonus. The arms were unaffected, except for a fine fibrillary tremor in the right. Nystagmus was observed, and he tended to wet himself, due, possibly, the observer thinks, to the fact that he was unable to move fast enough to answer an urgent call. Both the patients were bright and intelligent.

In the other family, two brothers were affected at the ages of 9 and 2. They were 12 and 13 years old at the time of examination and showed signs similar to those in the previous cases. They both exhibited great rigidity of the legs with exaggerated knee-jerks and ankle clonus. Nystagmus was not observed. Speech was stuttering. One had marked talipes equino-varus and the other dribbled incessantly, but was able to whistle. They were both mentally backward, though the more intelligent had been deaf since scarlet fever at the age of $3\frac{1}{2}$; both suffered from incontinence of fæces.

The cases I have now to consider are examples of what is known as hereditary cerebellar ataxy, a term introduced and applied by Marie [22] to a certain number of cases, that he collected from the literature, and which

he assumed owed their clinical signs to an inherited defect of the cerebellum. These cases were not seen by Marie personally, but he considered that they fell into a natural group on account of a certain similarity of clinical signs, and on the evidence of two *post-mortem* examinations.

Marie used for his paper two cases reported by Fraser [9], three by Sanger Brown [7], three by Nonne [31], and three by Klippel and Durante [18]. His idea was elaborated by Londe [21], who added besides others a case reported by Menzel (25). In this paper I have considered two other sets of cases which have been subsequently published by Neff [29] and Miura [28].

Marie considered "hereditary cerebellar ataxy" to be a disease characterised by an ataxy which develops later in life than Friedreich's ataxy, and which is consequently more often distinctly hereditary. The gait is of a reeling, drunken type, with difficulty in maintaining the erect attitude, and Romberg's sign is absent as a rule. There is increase of the tendon reflexes and occasionally ankle clonus can be obtained. The eyes are affected with irregular nystagmoid jerkings, the ocular muscles are frequently paralysed, and the pupils react imperfectly to light and accommodation. The visual field is often diminished, and there are changes in the optic discs. Speech is slow and explosive, there is an absence of paralytic club foot, and the sphincters are intact.

Nonne's cases [31] may be considered first. There were three brothers, aged 46, 49, 40 affected at the ages of 30, 14 and 10. They showed a progressive ataxy of the lower, then of the upper, limbs, without loss of muscular power and without contractions (though a peculiar difficulty in relaxing the muscles after a voluntary movement had been made is mentioned) without affection of sphincters, without affection of sensation, and without material alteration of tendon or skin reflexes. A peculiar nasal explosive speech was present in all three cases, accompanied in the first two by over-action of facial muscles during movements of expression. In all there was nystagmus and limitation of ocular movements; and optic atrophy existed in two cases.

One patient died, and at the autopsy there was found :— Smallness of the cerebellum, cerebrum, pons, medulla and cord : An excessive number of fine fibres in the posterior roots due either to atrophy or non-development of the large fibres : No degeneration of any tract in the spinal cord.

This *post mortem* induced Senator [37] who believed "hereditary cerebellar ataxy" to be allied to Friedreich's disease, to formulate the theory that the primary lesion in both cases is congenital non-development of the nerve-centres, and he considered his position strengthened by the publication of the autopsy on Menzel's patient, who, although clinically resembling Nonne's patient, showed on *post-mortem* examination, besides the smallness of the cerebellum, sclerosis of the posterior and lateral columns of the cord [25]. This man at 17 had difficulty in writing, at 28 difficulty in walking and in feeding himself. At the time of examination at 39, there was ataxy of all four extremities, an ataxic gait with jactitation of the feet and Romberg's sign, weakness of the right arm, rotation of head to the right due to spasm of sterno-mastoid, over-action of facial muscles, difficulty in relaxing his grip, increased knee-jerks, pupils large and sluggish in their reaction to light and accommodation, speech loud and explosive, sensation normal. Bernhardt [2], commenting on the cases of Nonne and Menzel in reference to their relation to Friedreich's ataxy, says "possibly the want of proper nervous development is the primary or hereditary factor; the spinal degeneration a special and engrafted feature, in virtue of which the case becomes relegated to one or other of the forms described by Friedreich or other observers."

In two other cases the sole *post-mortem* change was non-development of the cerebellum. One of these was reported by Fraser [9] and the other, a good example of "cerebellar ataxy," was recorded by Miura [28], from Japan.

In Fraser's case [9] there was reduction of the cortical grey substance of the cerebellum to little more than half the normal thickness, marked reduction in number of Purkinje's cells, which were shrunken and contorted, while

the cord showed no change. Attention has been called by Mackie Whyte [45] to the fact that when this case was examined, the only method of staining the spinal cord was by means of hæmatoxylin, so that it is not possible definitely to exclude the presence of sclerotic changes in the columns of the cord.

In Miura's case the cerebellum, pons, medulla and cord were greatly reduced in size, but no microscopic change of any kind was discovered.

That changes in the cerebellum are not always present in cases that Marie included in his group is shown by the autopsies recently made on some of Sanger Brown's [7] cases which were utilised by Marie. These cases are so important that they must be considered at some length. They occurred in a family where twenty-five members in four generations were attacked by a form of hereditary ataxy. Referring to the members of the family that he described Sanger Brown [7] mentions that they suffered from a disease which might be traced through several—at least four —generations, as it descended, tending to increase, to occur earlier, and to advance more rapidly. It usually attacked several members of the same family. It occurred most frequently between the ages of 16 and 35, but it might begin as early as 11, or as late as 45. An exciting cause could rarely be shown, but anything that depressed the general health might produce a rapid advance of the symptoms. There was always considerable inco-ordination of all the voluntary muscles and a sluggishness of the movements which these muscles produced. This was usually noted first in the muscles of the legs, but in a few months or years extended to the arms, face, eyes, and organs of speech. Ataxy was often extreme, and the gait devious, the patient deviating several feet on either side of the intended line of progression. The ataxy was not greatly increased by closing the eyes. The sense of position was perfect. Some weakness of the muscles of the legs without atrophy was frequently an advanced symptom, and occasionally there was permanent spastic contraction of the legs. In developed cases there were usually choreiform movements

of the head, and often of the arms, accompanying all voluntary movements. These irregular movements occurred in the hands, legs, or head, whenever the patient attempted to maintain either of these parts in a fixed position by a voluntary muscular effort. Movements ceased during sleep. There was usually some degree of static ptosis, with over-action of the levator on looking upwards. There was no nystagmus of any kind. Atrophy of the optic nerve was a constant and early symptom, and usually progressed slowly with the other symptoms. The response of the iris to light and accommodation was sluggish, and diminished with the advance of the optic nerve atrophy; when atrophy is complete, there may be absolute internal and external ophthalmoplegia. There was always marked disturbance of articulation, probably due to inco-ordination of the muscles concerned in speech. Occasionally the sphincters were slightly but definitely affected; but this symptom only appeared in those cases where spontaneous pains in the legs existed. There was no disturbance of sensibility, and no vaso-motor or trophic symptoms. The knee-jerks were always exaggerated, ankle clonus was frequently obtained, and the cutaneous reflexes were always exaggerated, but to a less degree. There was never paralytic club foot nor any other deformity, excepting rarely spastic contractions of the legs in advanced cases.

Three of these cases came to autopsy, one [26] showed no circumscribed cerebellar lesion, and no marked disease of Purkinje's cells. There was no obvious degeneration of any tracts in the cord, except those known to have cerebellar relations; that is to say, there was diffuse degeneration of the afferent cerebellar tracts, with special involvement of the direct cerebellar. The other two cases were being examined at the time of Patrick's paper [33], where they were mentioned, but, up till then, examination of the cerebellum had been negative, while there was marked degeneration of certain tracts in the cord.

A family reported by Neff [29] was affected with a very similar condition to that described by Sanger Brown. There were thirteen cases in four generations, but the onset was

very late, none of them being affected before the age of 50, and one not till 72. This case ran a long course and died at 84. Another family, the records of which I have not been able to see, is reported by Linnmalm [20], in which eight members out of a family of thirty-three were attacked by a gradually-progressing ataxy of all four limbs, with exaggeration of knee-jerks and ankle clonus.

Another of Marie's cases was reported by Klippel and Durante [18]. Three children, and their mother and maternal aunt being affected. The cases are very abnormal, being associated with lightning pains, gastric crises, and very marked alteration of sensation. The sister and brother came to autopsy [41]. In the sister, who died at the age of 48, the cerebellum was not affected, but changes were found in the cord. There was atrophy of gray matter, especially in the columns of Clarke and round the base of the anterior horns, together with degeneration of the posterior columns, chiefly the columns of Goll and degeneration of the tract of Gowers. The posterior roots were small, a condition due to the large number of small fibres present. In the case of the brother, who died after the age of 40, there was sclerosis of the columns of Goll and Burdach, the tract of Gowers, and the direct cerebellar tract. The posterior roots were intact. The pons was small, owing to diminution in number of fibres in the middle cerebellar peduncle. The number of convolutions of the cerebellum was diminished, and there were minute changes in the cortex.

I have mentioned on page 115 that Hodge noted wasting of the small muscles of the hand, and a condition of main en griffe in two of his patients, a condition we are familiar with in progressive muscular atrophy affecting the eighth cervical and first dorsal segment. Nolan [30] records wasting of the small muscles of the hand in two of his patients, and a brother and sister suffering from Friedreich's ataxy, examined by Mackie Whyte [45], exhibited main en griffe when an attempt was made to extend the fingers. Rook [34] noted wrist drop in one of his cases, and in another there was wrist drop with decided atrophic changes, and the "electrical changes were less than normal."

Two of Griffith's cases [14] had claw-like grasps. In some instances the atrophy is not limited to the small muscles of the hand, for Bramwell [5] records three cases of Friedreich's ataxy occurring in a family of nine. In one case, aged 21 at the time of examination, there had been complete paralysis of the legs for eleven years; in another case there was wasting of the muscles of the forearm and hand, and also of the legs. In the third case, that of a girl, aged 15 years, the muscles below the knee were atrophied, and there was also a paralytic ptosis. In another family reported by the same observer [3], two brothers and a sister, afflicted with Friedreich's ataxy, exhibited a condition of main en griffe with wasting of the thenar and hypothenar eminences, and all three showed atrophy of the flexor muscles of the feet. A daughter of the elder brother also showed a condition of main en griffe.

Wilson [44] notes muscular atrophy in two children under his care, associated with diminution of reaction to faradism and general diminution to galvanism, with A.C.C. greater than K.C.C.

In Smith's [40] cases, muscular atrophy was present to a marked degree. In one patient, aged 37, in which the disease had started at 6, there was complete muscular paralysis of the lower limbs, and only the slightest possible movement in the hand. In another case of twenty years' standing which came to autopsy there was complete paralysis of legs below the knee, with wrist drop, and an inability to raise the arm above the level of the shoulder.

Mackay [23] records that, in his case of Friedreich's disease which came to autopsy, there was wasting of all the limbs; but, although the reaction of degeneration was not present, yet there was diminution of muscular irritability to both the galvanic and faradic currents. He also mentions that the fingers of this patient were generally hyperextended at the metacarpophalangeal joints, and flexed at the interphalangeal. Two sisters of this patient, also suffering from the disease, showed a tendency to main en griffe. This observer [24] has collected the pathological findings of eighteen undoubted cases of Friedreich's disease, and of

these eighteen cases the condition of the anterior horn cells is noted in eleven, and changes were present in six.

In his own case there was "diminution in the volume of the anterior horn cells." In Smith's cases quoted above, the "anterior horn cells were less numerous and much altered." In one of Friedreich's cases [11] "the ganglion cells were fewer than normal." In Burr's [8] patient there was "atrophy of a few cells in the anterior horn." In Mirto's [27] "thinning of the nervous reticulum with atrophy of a few cells," and in Simon's [38] there was a "diminution in the number and volume of the cells."

Thus there seems to be clinical and pathological evidence that muscular atrophy, due to involvement of the anterior horn cells, may occasionally be present together with the signs of Friedreich's disease. And in this connection two families, one reported by Gee [12], and the other by Ormerod [32], are of exceptional interest.

In the family reported by Gee, the father, aged 37, was never able to walk properly, and tenotomy had been performed at 19; subsequently, both his great toes were amputated for perforating ulcers. His arms were fairly nourished, and the deltoids were large, the small muscles of the hand were wasted, though the movements and grasps remained good; reaction to faradism was said to be normal, but absent to galvanism. Sensation was unimpaired. His gait was spastic, and he was unable to walk without crutches; there was much spasm of the adductors, and he was only just able to move his ankles; his legs were rather small. Thigh clonus was obtained on tapping the patellar tendon, though ankle clonus was not elicited.

Two of his children, aged 12 and 11, seemed similarly affected. In both, difficulty in walking made its appearance in early childhood. At the time of examination they both used crutches; there was much rigidity of the legs. The knee-jerks were excessive, and the feet valgus. There was no alteration of sensation, and the sphincters were normal. In the elder child the hands were normal, but possibly showed some wasting of small muscles. In the other the muscles of the shoulders were well developed, the arms were

small, but not wasted, and there was some slight rigidity; the wrist and elbow jerks were excessive, and the thumbs could not be abducted. Sensation was good, and all the muscles reacted to faradism.

In the family reported by Ormerod, three brothers were affected with the same disease, though to a variable extent. The eldest, aged 23, had had difficulty in walking for seventeen years. At the time of examination the legs were held rather stiffly, there was but little movement at the ankles, and the toes did not leave the ground in walking, the knee-jerks were much increased, ankle clonus was present, and the plantar reflex was of the extensor type (Babinski). There was uniform wasting of the muscles below the knee, which had only been noticed three years, but the electrical reactions were normal. There was atrophy and loss of power in the short muscles of the feet, and in these no electrical reactions were obtained. Wasting and loss of power of four months' duration were present in the small muscles of the hands, more marked in the right. Tendon reflexes were well marked. Electrical reactions were normal, except for total loss of response in the thenar and first interosseous muscles of the right hand. There was no sphincter trouble, sensation was not affected, and the pupils reacted normally. Nystagmus was present, and there was no optic atrophy.

The next brother, $3\frac{3}{4}$ years old, showed some difficulty in walking. He walked on a wide base, balanced with his arms, and his feet seemed to stick to the ground. The plantar reflexes were of the extensor type, the knee-jerks exaggerated, but ankle clonus was not obtained. His mother stated he was able to walk quite naturally till ten months previously. Nothing else abnormal was noticed, except an antero-posterior lumbar curvature, and some weakness of electrical reactions of the extensor muscles of wrist and fingers on both sides.

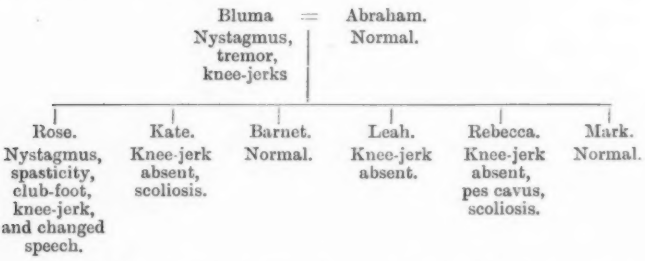
A third brother, aged 7, showed merely some unsteadiness in turning, exaggerated knee-jerks and Babinski's sign.

These two families show the occurrence of muscular wasting associated with spasticity, and if we turn to the account of a family reported by Herringham [15], we find

muscular atrophy, associated in one case at any rate with the reaction of degeneration, in twenty-one cases in five generations of one family. As a rule the atrophy began in the peronal muscles, and generally affected the tibial group. Occasionally the calf muscles were subsequently attacked, and in some cases the small muscles of the hands. In this family it is noteworthy that in all cases the disease was transmitted through the female members, who remained unaffected.

I now come to the consideration of a family I have had the opportunity of examining through the kindness of Dr. Henry Head, who has one of the children under his care at the London Hospital. I am also indebted to Mr. Mayou for his assistance in the examination of the eyes of these patients.

In this family, the mother and the eldest daughter are affected with the same disease which is more advanced in the case of the daughter. She resembles the cases I have already quoted in that she is an example of a spastic paraplegia, but in addition she exhibits tremor on voluntary movement, nystagmus on voluntary ocular movement, and both feet are in a condition of pes cavus. Three sisters, apparently quite well, have no knee-jerks.



The mother, *Bluma Abrahams*, a Russian Jewess, came to England with her husband and family in 1894. She is an undersized, but healthy, woman, aged 38. There is no history obtainable of any instance of paralysis, club foot, or tremor, occurring in any member of her family. Her hus-

band, aged 43, is a well-built, strong man, temperate, with a good family history. She has had six children, but no miscarriages; her health during all her pregnancies was good. All the children were full term, and she suckled all of them for twelve months, except the youngest, aged 2, who was brought up by hand; all her confinements were uneventful, and instruments were not used in any of them. All her children are alive, namely, Rose 17, Kate 14, Barnet 9, Leah 7, Rebecca 5, Mark 2.

She complains of a tremor of the hands, which she has noticed for some years, but is unable to give any idea as to when it first appeared, but she noticed that it came worse after the birth of Rebecca, five years ago. She is certain that it was not present when she came to England ten years ago.

On examination, the nervous system alone is found affected, in the following particulars. She has a tremor of the hands, which is only noticeable on voluntary movement. The tremor is fine and rapid, and resembles that seen in disseminated sclerosis; it is well brought out on asking her to pick up a pin, and it interferes with the performance of fine movements, so that she is unable to thread a needle. Her gait is not affected, she can stand quite steadily with her eyes closed. Her feet are large and well formed, and there is no rigidity. All her tendon reflexes are brisk, her knee-jerks are exaggerated, and her elbow and wrist-jerks are somewhat glib. Ankle clonus cannot be obtained, and, though attempts have been made to elicit the plantar reflex on several occasions, no response has ever been obtained. No abnormality in the movement of the eyes can be detected, unless she looks to the extreme right or left, when a well-marked horizontal nystagmus is apparent; this nystagmus is also brought out if she fixes for a point held just above the level of her eyes and about five inches away, and it is then noticed that the frontalis muscle contracts synchronously with the movements of the nystagmus. She was quite unaware herself that her eyes were affected in any way. Her vision has been failing for some few years, but with glasses (left—2D. right—3D.)

she is able to see $\frac{6}{12}$ with each eye. Her fundi appear normal, and there is no paresis of any of the ocular muscles, and no diplopia. There is no interference with the functions of the bladder or rectum.

Rose Abrahams, aged 17, the eldest daughter of Bluma Abrahams, was born within a year of the marriage of her parents. She has been affected with a gradually-progressive difficulty in walking since early childhood, with a deformity of the feet which was first noticed about the age of 3, with a tremor on voluntary movement and nystagmus; both of these were noticed when she was in Russia ten years ago, but I can obtain no account of the approximate date of their first appearance. Nystagmus and tremor on voluntary movement were obviously well developed when she was 7, for at that age she went to the Board School in London, and the other children used to laugh at her for trembling and jerking her eyes about.

She was always a clumsy child, and she did not walk at all till she was 3 years old. Mentally she is quite bright, and although she knew no English when she was sent to the Board School, at the age of 7, yet she managed to reach the fifth standard by the time she left at 14.

At the age of 1 she had whooping cough, and towards the end of the illness she had some kind of convulsive attack; according to the mother's account, she had an attack of convulsions after a fit of coughing, involving all the limbs, in which she became unconscious and went very blue. She remained unconscious for some considerable time, and the convulsions were repeated at intervals. This seizure seems to have been the climax of the whooping cough, for after it she made a rapid recovery. At the age of 2 she had diphtheria, but only slightly, and she does not seem to have suffered from paralysis. She has had no other illness, except measles at the age of 5.

Her articulation is altered in a remarkable manner; thus, even when she was an inmate of the London Hospital, her articulation seemed sometimes to be affected, and sometimes not. In January, 1904, I made a note that there was no alteration to be noticed in her speech, but shortly

afterwards noted that she had a difficulty in enunciating certain words. Subsequently I have seen her on several occasions, when neither I nor other observers have been able to detect any abnormality whatever. She herself says that she has recently had to talk much more slowly than she did, and has to be much more careful over long words. On the days when any alteration is perceptible she speaks slowly and hesitatingly, and pronounces all her words very deliberately.

She frequently gets up in the morning with a headache, but it almost invariably passes off in the course of the day. She has never had any fit or other attack since the convulsion during the time she suffered from whooping cough.

Her gait is typically spastic. She walks with stiff legs and swings them forward from the hips, inclining the body to the opposite side to enable the toes to clear the ground, but, nevertheless, the feet catch at every step, and her boots are worn through at the toes and over the ball of the foot. She is able to walk quite rapidly, taking little, short steps, and she is in no way unsteady. She never feels giddy and she has never fallen. When she wishes to turn quickly, she stands on one leg and twists her body rapidly into the required position. It is obvious that she would be unable to accomplish this manœuvre if she suffered from any degree of ataxy.

There is no wasting of any group of muscles in the legs, and muscular power is good, but there is considerable spasticity, which is more marked on the left side. Her arms show no sign of wasting, and the grasps are good; there is no rigidity, and no difficulty in relaxing her muscles after a voluntary movement has been made. They are affected, however, with a very marked intention tremor, more violent than her mother's, and more marked on right side. The hands, as they lie in her lap, are kept perfectly still, but on attempting to move them they are seized with a most pronounced tremor. Her writing is profoundly affected.

She is unable to thread a needle unless it has a very large eye, and she is almost unable to pick up a pin; in

attempting to do so, she brings her hand rapidly to where it is lying and then fumbles at it till she picks it up, the tremor increasing the nearer the hand gets to the pin. She does not show the "hovering" "bird of prey" movement described by Charcot, which is so repeatedly mentioned as occurring in some cases of Friedreich's ataxy. There are no swaying movements of the body and head when at rest.

There seems to be no inco-ordination at all; she is perfectly able to walk along a straight line without deviating from it. She is unsteady when she stands with her feet together, but this is accounted for by the deformity of her feet. She does not tend to fall with her eyes closed, though her unsteadiness is perhaps a little increased. She is able to touch any part of one leg with the toes of the other foot. With regard to her hands, she can bring her two index fingers together accurately and touch the tip of her nose with the eyes open or closed, but these movements, being voluntary, are associated with considerable tremor, which makes them rather difficult of performance. She has no loss of the sense of position; she knows the position of her limbs when they are passively moved about whilst her eyes are closed. There is no alteration of sensation to touch, pain, heat or cold in any part of the body. The superficial reflexes are all present. The plantar is present, and is of a characteristic extensor type (Babinski's sign). The knee-jerks are very exaggerated, but equal on the two sides, and if the patellar tendon is tapped while she is lying in bed, a thigh clonus is obtained. Ankle clonus is well marked, and the elbow and wrist jerks are very brisk. The jaw jerk can be obtained, and is not exaggerated. Vision has always been defective; without glasses she is able to see $\frac{6}{18}$ with the left eye and $\frac{6}{60}$ with the right.

Under atropine with $\frac{=3\text{D. spherical}}{=5\text{ cylinder ax. }60^\circ}$ she can see $\frac{6}{36}$ with the right, and with $\frac{=\cdot75\text{D. spherical}}{=\cdot25\text{ cylinder Ax. }60^\circ}$ she can see $\frac{6}{18}$ with the left eye. There is some slight contraction of the visual field, as shown by the perimeter chart, and the disc and fundus are normal in both eyes.

Smell, taste, and hearing are all normal. There is no

ptosis, no diplopia, and no paresis of any ocular muscles. Her eyes are in a state of constant fine horizontal movement, which is more marked if she fixes or turns her eyes to the extreme right or left. The movements of the right eye are much more marked than those of the left. The pupils are equal, and react to light and accommodation. There is no facial paralysis, the palate and larynx are normal, the tongue is protruded in a straight line and is tremulous.

Catamenia began at the age of 13, but the flow is scanty and irregular. The functions of the sphincters are not affected in any way.

There is a slight lateral curvature of the spine in the dorsal region, with the convexity to the left. The feet are in a position of marked pes cavus, the heels are drawn up and the soles of the feet look inwards, so that there is a condition of talipes equino-varus as well. The feet are much shortened from before backwards, with exaggeration of the plantar arch; the toes, especially the great toes, are extended on the metatarsus, and the terminal phalanges of all are flexed. The deformity is more advanced on the left side.

Of the other children, the second child, *Kate*, is in every way normal, except that no knee-jerk can be obtained on either side, even with reinforcement. There is no nystagmus or tremor; her feet are normal.

Barnet, the third child, is in every way normal, and in him the knee-jerks can be obtained even without reinforcement.

Except that no knee-jerks can be obtained, even with reinforcement, the fourth child, *Leah*, also shows no sign of disease.

In the case of *Rebecca*, the fifth child, no trace of a knee-jerk can be obtained even with reinforcement. Her feet show an early condition of pes cavus. They are shortened from before backwards with exaggeration of the plantar arch, and the great toes sometimes straight are usually hyperextended on the metatarsus. The deformity of the foot is not permanent, and it can be corrected by passive

movement. The plantar reflex is obtained, but it is difficult to be certain of its form, as sometimes the great toes are strongly extended and sometimes flexed. Vision is defective, and is only slightly improved by glasses. Under atropine with $\frac{-2{\cdot}25\text{D. spherical}}{-4{\cdot}25\text{ cylinder ax. vert.}}$ she can see $\frac{6}{24}$ with the left eye, and with $\frac{-2\text{D. spherical}}{-5{\cdot}5\text{ cylinder ax. vert.}}$ she can see $\frac{6}{24}$ with the right. She has a well-marked divergent strabismus which disappears for an instant when she fixes for near vision, but the eyes almost immediately diverge. This strabismus cannot be due to the hypermetropia as divergent squint is unknown in that condition, and it is not due to amblyopia of one eye, as she can see $\frac{6}{24}$ with each, and so by a process of exclusion it appears that this squint is due to a paresis of the internal recti. The optic discs show no trace of atrophy or any other change.

The youngest child, *Mark*, aged 2 years, seems to be quite unaffected, and his knee-jerks can be obtained with ease.

It may be mentioned that while all the girls show a strong family likeness to each other, and to their mother, the boy, Barnet, in no way resembles his sister, but conforms to the image of his father.

Additional Note [*March*, 1906].

I have recently had an opportunity of examining this family again. No new symptoms have developed in the case of Rose, though she is definitely weaker, and has to spend most of her time sitting in a chair.

Kate has to wear a spinal jacket for a scoliosis, which has rapidly developed in the last two years, and a similar condition is just making its appearance in the youngest girl, Rebecca.

The cases I have considered in this paper are examples of some of the many forms in which family diseases of the nervous system can occur, and I think that they, together with the definite type known as Friedreich's ataxy, can be looked upon as different manifestations of the same disease. One might almost call them different varieties of the same species.

The common form of Friedreich's ataxy passes almost imperceptibly into the form of spastic paraplegia described by Bayley. In Friedreich's ataxy the clinical signs due to sclerosis of the posterior columns of the spinal cord overshadow those due to the sclerosis of the lateral columns, and the absence of the knee-jerk is characteristic. But there are some families, as I have shown, in which, while some members are affected with what is undoubtedly Friedreich's ataxy, others, though clinically resembling them in all other respects, differ only in possessing a knee-jerk. In some more distinct cases all the affected members of a family have knee-jerks, while in other instances not only are the knee-jerks retained, but are even exaggerated, and ankle clonus can be obtained in addition. Thus the signs of sclerosis of the lateral columns gradually becomes more apparent, and the cases reported by Bayley are examples of a simple lateral column sclerosis occurring as a family disease without any sign of involvement of the posterior columns at all.

Tooth's cases resemble Bayley's in the main manifestations, but are not of such a pure type, as some show nystagmus, speech defect, and involvement of the sphincters, while two of them exhibit mental deficiency, which is also a complication of Nolan's cases, and which seems to be fairly frequent in many cases of "cerebellar ataxy."

The cases grouped together as "hereditary cerebellar ataxy" comprise a heterogeneous group which will have to be subdivided on further investigation; but at present they may be looked upon as examples of a family disease which is characterised by inco-ordination affecting all voluntary movements associated with increased tendon reflexes and ankle clonus, while in addition some families—such as the one described by Sanger Brown—exhibit a tremor affecting voluntary movement. Further, many cases show optic atrophy, with oculo-pupillary changes and paresis of the ocular muscles. But deformities of the feet are rarely if ever seen. My own case of Rose Abrahams is peculiar in many ways. She is an example of an hereditary disease, for her mother is similarly, though less, affected. She conforms to the type of disease described by Bayley, as she is an

example of a spastic paraplegia without any inco-ordination. But she differs from that type in that she shows an involvement of the centres concerned in the initiation of voluntary movement, as is shown by the tremor affecting the arms, speech and ocular movements; in this she approaches Sanger Brown's cases, though the tremor in her case resembles that seen in disseminated sclerosis, while in his cases it is of a choreiform nature. She shows some relation to Friedreich's ataxy in possessing a typical pes cavus, and in this respect differs from the group of cerebellar ataxy, when this deformity is almost unknown, and, further, she again differs from these cases in the entire absence of ocular paresis and optic atrophy.

Perhaps the most interesting feature about this family is that, while the eldest child—Rose—is such a definite example of a spastic paraplegia, yet in her three sisters there is a complete absence of the knee-jerks, and the youngest girl seems to be a very early example of true Friedreich's ataxy; though the paresis of the internal recti noted in this case points to a condition not usually associated with this disease.

Considering the question of the relation of "cerebellar ataxy" to other forms of family nervous disease from the pathological side, it is worthy of note that, of all the cases of "cerebellar ataxy" that have come to autopsy, changes limited to the cerebellum have only been observed in three, namely, those of Fraser, Nonne, and Miura, and all the others have shown definite degeneration of certain tracts in the spinal cord, and I have already mentioned that Senator and Bernhardt have stated their conviction that in these family diseases of the nervous system, non-development of the nervous centres may be the primary condition in which secondary degenerations are prone to occur.

Londe even goes so far as to say that "cerebellar ataxy" is a definite entity, and is allied to Friedreich's ataxy, for in both there is degeneration of the cerebro-spinal system, which begins in the spinal cord in Friedreich's ataxy, and in the cerebellum in "cerebellar ataxy," which, in some cases, such as the one described by Menzel, there

is a mixed degeneration affecting both the cord and the cerebellum.

Very frequently the clinical signs of any one particular case of family nervous disease may be roughly divided into two classes, on the one hand, there are the main features, whereby the case is relegated to its position in some definite group, and, on the other hand, there are minor features which are of extreme importance, and which I look upon as forming connecting links, as it were, whereby it is sometimes possible to show relations between the case in question and cases in other groups. For instance, the frequent occurrence of muscular atrophy in cases of Friedreich's ataxy and in some of the other forms of family nervous disease which I have considered in this paper seems to show some, though perhaps distant, relation between these cases and those recorded by Herringham, where muscular atrophy involving definite groups of muscles occurred in twenty-one members of one family.

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NOTE ON APPARENT RE-REPRESENTATION IN THE CEREBRAL CORTEX OF THE TYPE OF SENSORY REPRESENTATION AS IT EXISTS IN THE SPINAL CORD.

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THE first part of the subject we wish to discuss in the present note is necessarily what differentation exists between the cortico-cerebral type and the spinal root type respectively of the representation of tactility, *i.e.*, the sensation of touch.

Now, as regards the representation of sensation in the cortex cerebri, the majority of neurologists hold that the kinæsthetic, or excitable region of the cortex, is the seat of representation of sensation as well as motion. There remain two other schools of thought, one claiming that while representation of tactility occurs in the excito-motor cortex, there is also another field, or perhaps more than one field, of the cortex in which the same function is located, and finally there is a relatively smaller school who do not believe that the sensory-motor area is in any way the seat of sensory representation.

We will, therefore, briefly review the three classes of views on sensory representation in the pallium so far as they relate to the kinds of sensation represented, for although one of us has for many years shown grounds for believing the type of cortical sensory representation to be segmental[1] in

[1] In this connection the word segment means the regional divisions of the body, *e.g.*, for the upper limb, the phalanges, metacarpus, carpus, forearm, arm, &c.

character, the question does not find (except in Dercum's American work) full discussion in neurological text-books.

(1) Representation of Sensation of any kind in the Pallial Kinæsthetic Area.

Hitzig [1] was the first to prove that lesions of the so-called motor cortex caused loss of muscular sense. This was confirmed subsequently by Tripier [2], Moelli [3], Luciani and Sepilli [4], Munk [5], Bechterew [6] (quoting his later opinions), and Mott [7]. Of these the most direct experiments were those by Mott upon monkeys. From the effects of the lesions which he made in the kinæsthetic region of the monkey's brain, it distinctly appeared that both the tactile sense and pressure sense were represented in the so-called motor region. At the same time it is to be observed that all the foregoing investigations were made before the topographical anatomical researches of Campbell, and later of Brodmann, and that no precise distinction was made between the pre- and post-Rolandic gyri which together constitute the Rolandic zone of authors. Consequently the experimental results at present available will require reinvestigation at some time to secure a correct comparison between structure and function, but, so far as contrast between sensory representation in the Rolandic zone and that in other parts of the pallium is concerned, the facts obtained are of the highest value. The primal difficulty in solving this problem is, of course, the extreme rarity of strictly localised lesions of the cortex in man, but as attention is now concentrated on the point, it may justly be hoped that this hiatus will be filled by more clinical work in this direction.

Against these numerous positive results must be quoted the negative experiments of Ferrier [8], those of Schäfer [19], to whose discussion of the subject special reference should be made, and the earlier experiments of Schäfer and Horsley [9]. As regards the investigations of the latter authors, they were made in association with other experiments on the gyrus fornicatus, and

related, not to the muscular sense, but to tactile æsthesia and algesia. The negative character of these experiments of course deprives them of much of their importance, and the fact that Schäfer and Horsley did not observe defects of tactility in their lesions of the Rolandic zone cannot be held to weigh against the enormous mass of evidence which has accumulated from clinical and further experimental work, and which establishes the fact that in the Rolandic zone tactile representation is present, though to a limited degree. (*cf.*, Mills [11], Walton and Paul [19], &c.)

(2) Representation of Tactile Sensation elsewhere in the Pallium as well as in the Kinæsthetic Cortex.

All observers are agreed that isolated lesions of the Rolandic zone do not produce complete tactile anæsthesia or analgesia,[1] and have reasonably assumed therefore that however definite the representation therein of the muscular sense may be, at any rate that of tactility and pain must be additionally represented in some other part of the cortex cerebri. The researches of Ferrier and Schäfer and Horsley seemed to show that the limbic lobe in whole or in part was this alternative or additional seat of sensory representation, but here again much further critical research is needed before this region of the pallium can be accepted as part of the tactility sensory area, especially in view of Campbell's [12] demonstration of the differential structure of the cortical regions. At the present time not only is it impossible closely to criticise the facts upon which the view of multiple representation of tactility is based, but it also must not be forgotten that we have no means as yet of absolutely differentiating between a response which may directly emanate from the ventral nuclei of the optic thalamus and that which results from thalamic excitation of pallial centres.

[1] To these must also be added temperature anæsthesia.

Of course when the attention of the person observed is concentrated on a sensory stimulus, the response thereto must be pallial in origin, but a pallial response does not necessarily involve the coincident excitation of consciousness.

(3) No Representation of Sensation in the Kinæsthetic Cortex.

Reference must now be briefly made to the view that the excito-motor cortex is only motor in function and subserves no representation of sensation. This view has always appeared to us to be contrary to the evolution of the nervous system, since from the point of view of function every part of the central projection apparatus must be sensori-motor, and from the morphological standpoint all the homologies of the structure of the pallium compared to that of the lower nerve centres, establish the same principle.

Evidence bearing on the subject from Clinical Observation.

During the past decade an admirable monograph on the clinical investigation of the subject of conduction of sensation in the central nervous system has been issued by Dr. Long, but it is certainly necessary that a fresh critical examination should be made of a large series of cases of localised lesions in the Rolandic zone, to determine the true correspondence between the area of cortex destroyed and the correlated part of the body in which loss of sensation consequently appears. However, some definite results have already been obtained in this field.

Thus Horsley [10] has shown, since 1886, that in cases in which destruction or injury of the kinæsthetic cortex has caused motor defect in certain parts, there is a correspondingly localised sensory loss, secondly, that there is a topographical identity between the parts affected with motor and sensory paralysis respectively, and, thirdly, that

the loss of sensation is roughly parallel in degree to the loss of movement.

This correlation between movement and sensation suggests, of course, that the representation of these two intimately-associated functions is localised in one and the same area of cortex. But at the present time there is no clinical evidence confirmed by post-mortem examination which can be said definitely to invalidate the view put forward by Mills [11] and Campbell [12][1] that the post-central region is essentially the seat of sensory (? tactile) representation, and that all the instances of apparent lesion of the ascending frontal gyrus only are probably vitiated by associated disorder of the ascending parietal convolution, and finally that the corresponding "sensory" centres are arranged horizontally behind the "motor" centres.

Accepting provisionally that in this class of case the part lesioned is the Rolandic zone, *i.e.*, both central gyri, then the kinds of sensation disturbed Horsley proved in 1885-6 from clinical observation to be: (1) slight tactility; (2) topognosis; (3) general orientation of the paretic part, and occasionally (4) pain.

Further, that the disturbance of tactility and topognosis was such that in all cases direction of the localisation error caused by defective cerebral cortical representation of sensation was propinquity or proximality, *i.e.*, the point indicated was nearer the body than that actually stimulated. The error was also shown to be segmental in character, and its proximality varied directly with the extent of the lesion, so that in a severe or extensive case the patient when touched lightly on a terminal phalanx indicated a spot on the dorsum of the metacarpus, and in a less severe case on the next phalanx, or next phalanx but one.

These facts during the past twenty years have proved in a very large series of cases to be correct, and have been

[1] It is evident from a study of Campbell's magnificent monograph that Horsley's views of the pallial representation of musculo-arthrodial and tactile sensation were not before him in discussing the question, *e.g.*, p. 97, *et seq.*

finally established by the control of diagnosis confirmed by operation or autopsy.

In addition to the foregoing disturbances of sensation, reference must be made to the stereognostic sense which can practically be regarded as a special function, although it is, of course, at present tested as a combination of the tactile and muscular sense of the palmar surface of the hand, and for this reason Claparede (1898, quoted by Long) is more correct in speaking, not of a stereognostic sense, but of stereognostic perception. As it is compounded, as just stated of those forms of sensation which are perturbed by lesions of the Rolandic zone, it is probably represented in that same part of the pallium, and, above all, in the arm area.[1]

Hence extensive lesions of this region of the cortex are always accompanied by astereognosis.

Thus up to the present time observation of the tactile anæsthesia and topognostic defects evoked by cerebral cortical lesions has shown that the type of their representation is fundamentally segmental in character, and not only is this true of organic lesions, as already described, but we desire to point out also that the original descriptions of Charcot of the sleeve, gauntlet and stocking anæsthesia in functional cases indicated a type of the same kind, namely, segmental.

Such segmental representation obviously has no relation to the anatomy of the peripheral nerves of the body nor any relation to the ideal metameric segmentation of the body. It is conceivable that we would be justified in regarding it as a natural arrangement of the memories of movements which themselves are necessarily segmental, and are always translating the parts of the body through space in varying degrees of proximity, or the reverse to the surface of the head and trunk.

[1] A careful map of the normal extent and limitations of the stereognostic sense of the whole skin and mucous membrane surface would be a very desirable addition to our means of diagnosis.

See also F. Krause's interesting results after cortex extirpations, *Deutsche Klinik*, 1904. Probably, however, his method of estimating tactility was not sufficiently delicate to exclude pressure.

We now wish to draw attention to an additional type according to which tactility and topognosis appear to be also represented in the cortex cerebri, viz., a type which is apparently a re-representation of that according to which tactility is represented in the spinal cord. The representation of tactility in the spinal cord, which is essentially a metamerically-divided organ, is readily mapped out, and the terminology descriptive of the topographical mapping of tactility in at any rate the hand and forearm was originally laid down by Ross [13] from clinical observations and by Paterson [14] from consideration of embryological anatomy, while since that preliminary epoch the subject of spinal sensory representation has been exhaustively examined by Sherrington [15] and by Head [16] with the result that the topographical areas of the different spinal roots are now well defined [17].

Confining ourselves in the present paper to the peripheral half of the fore-limb, we must imagine the human embryo placed in the typical vertebrate position, viz., with the axis of the spinal cord horizontal and the axes of the simple budding limbs at right angles to this. Under these circumstances it follows that the index, thumb and radial side of the forearm is pre-axial and the ulnar half post-axial, and these terms as originally introduced by Ross and Paterson have proved to be of practical utility. The pre-axial region is supplied by the higher metameres, viz., the fifth, sixth and seventh cervical, and the post-axial by the eighth cervical and first and second dorsal. As regards the convenience of subdividing the surface of the limb, the middle dividing line which should be theoretically the projection on the skin of the central axis running out through the middle finger, is on the contrary part of the field of the eighth nerve root or segment. We shall return to this later, but mention it here to emphasise the fact that the central line or axis of the limb would seem to have a special morphological significance and, as we shall subsequently show, it also has a clinical significance, which finds expression in the existence of a definite functional representation in the cortex cerebri.

Apparent Re-representation of the Spinal Cord Metameric Type of Sensory Topography in the Cerebral Pallium.

(*a*) *Contrast between post-axial and pre-axial regions of the limb.*

In the spinal cord the tactile sensory representation of the periphery of the upper limb is in arrangement essentially parallel to the central axis of the limb, and of course as we proceed from the upper level of the cervical enlargement to the lower end, we pass from the representation of the pre-axial side to that of the post-axial side. This must now be viewed in another way, *i.e.*, the relation of the axis of the fore-limb to the mesial sagittal plane of the body. It follows that if the forearm be parallel to the longitudinal axis of the body and the limb in the position of easy pronation, and if there be an error of conscious perception the question for decision will be whether the mistake in movement is in going towards or from the mesial sagittal plane of the body, whether the misjudgment is in fact pre-axial or post-axial in direction as far as the limb itself is concerned.[1]

Being accustomed to observe disturbances of the proximal position [18] (psychical localisation by the patient) of the segments in the fore-limb in cortical lesions it was in 1894 a matter of considerable surprise to one of us when examining a case of destructive disease affecting the area of the middle cerebral artery, to find there was present marked dissociation between the pre-axial and post-axial reaction to light touches as well as the ordinary (cortical) segmental tactile hypæsthesia and atopognosis. The patient, who was a highly-trained scientific observer, had himself previously recognised a certain sense of loss of relative knowledge of the sides of the fore-limb. This observation was mistakenly regarded as exceptional and

[1] The general cerebral representation of the relations of the body surface to the planes of space, especially the mesial sagittal plane, is partly discussed by Horsley in the "Boyle Lecture," 1905, and by himself and Slinger in the present number of *Brain*.

was not followed up. Recently, however, a further consideration of our precise knowledge of position in space has rendered it necessary to re-examine the topographical knowledge of the hand and fore-limb in cases of known lesions of the kinæsthetic cortex, one of us (Russel) therefore undertook this work and carried out a series of observations on a certain number of cases as they passed through the surgical department of the National Hospital, Queen Square. To the hospital cases are added two more observed in private practice.

In all, the lesion was in the Rolandic zone, and in all there was present in the contralateral forearm and hand (*a*) slight loss of tactility; and (*b*) marked atopognosis, of the segmental type, *i.e.*, in which the error, when the stimulus was felt at all, was proximal, the spot indicated by the other hand of the "blind-folded"[1] patient being from one to three segments nearer the body than that actually touched. Where the condition of the pathologically altered limb admitted of its use as an indicator the topognostic sense of the "normal" hand was tested. In each case it was unchanged, *i.e.*, the sensory localisation was normal.

Without prejudice to any interpretation of the meaning of the direction of the error, *i.e.*, to or from the mesial sagittal plane, the cases were examined for axial dissociation with the forearm in a position of easy pronation,[2] the hand being horizontal, at the level of about the ensiform junction and lying approximately in the sagittal plane passing through the nipple line.

The results obtained may be conveniently tabulated in abstract as follows:—

[1] It is always well in making observations on the appreciation of sensory stimuli not actually to blindfold a patient, nor to ask them to keep the eyelids cleosd, as these conditions occupy, however slightly, the attention. A screen held between the face and the hands answers far better than either of the other procedures.

[2] It had already been found by one of us (V. H.) that the delicacy of topognostic æsthesia on the palmar surface of the hand is so great that very little or no error can be detected in cases in which the segmental error is nevertheless extremely marked when the dorsum of the hand is the region stimulated.

TABLE OF RESULTS.

Case and Lesion found at Operation[1] or at Autopsy.	Abstract of Sensory Disturbance and Errors of Localisation.
Case 1.—G. M., male, 42 years [Dr. Beevor]. *Operation*, November 7, 1905. Pachymeningitis involving the right cortex over an area posterior to the fissure of Rolando and above the Sylvian fissure. This area included the lower half of the ascending parietal gyrus and the whole supramarginal gyrus. The postero-inferior angle of the part removed included both sides of the posterior third of Sylvian fissure. The depth of the incision was about 2 cm., *i.e.*, the floor of the lesion was composed of corona radiata (not softened).	(*A*) *Before operation.*—Slight diminution of sensibility to pain over hand and forearm to elbow, to camel-hair touch on fingers of left hand. Loss of sense of position in fourth and fifth fingers, marked astereognosis.[2] Post-axial error of localisation on dorsum of middle, ring, and little fingers of left hand. On the palmar surface there was no error. On the dorsum of hand and wrist the topognostic error was proximal. The errors were always much greater after a fit, and gradually diminished during the succeeding ten to fourteen days. (*B*) *After operation.*—(1) Immediately after operation, complete hemianæsthesia of tactility and thermal sense, hemianalgesia of face, upper limb and trunk, relative on lower limb, hemianopsia, hemimotorparalysis of arm and face, slightly of leg. (2) Fourteen days after operation, analgesia less marked. Visual fields restored; shoulder and elbow movements restored, also slight power of flexion and extension of the wrist and fingers. Complete loss of sense of position of the upper limb. (3) Twenty-eight days after operation. Returning post-axial estimation of position of hand.
Case 2.—M. B., 40 years [Dr. Risien Russell]. *Operation.* Glioma (mass removed 5 cm. longitudinal axis, 4 cm. frontal axis, 3 cm. vertical depth), situated immediately sub-cortical to the junction of the leg and arm centres in left hemisphere. The lesion left by the removal of the growth involved both the central ascending gyri; and extended into the corona radiata to a depth of from 3 to 5 cm.	(*A*) *Before operation.*—Soon after a Jacksonian fit, beginning in the foot and involving whole side and limbs, there was present in the right hand definite atopognosis (proximal error) and astereognosis, hemiparesis of right side. (*B*) *After operation.*—(1) Thirteen weeks after operation. Marked post-axial localisation of all light touches on dorsum of digits, as well as topognostic proximal error. (2) Sixteen weeks after operation. No analgesia, no loss of pain or temperature sense. Defective sense of light touches on dorsum of digits, normal on forearm, very marked proximal atopognostic error on digits and distal error on dorsum of carpus and metacarpus. The error was pre-axial on the fourth and fifth digits, and post-axial on the first and second digits. When tested with Horsley's divided plate[3] the post-axial error between the two index fingers was extremely marked.

[1] We do not, of course, desire to claim that the estimated position of a lesion exposed in an operation is necessarily correct, but offer the facts according to our observation at the time.

[2] In all the cases astereognosis was estimated in the usual way, *i.e.*, with coins, pencil, knife, watch, &c., and in all the palmar surface was tested; whereas (as before stated) for topognosis the dorsum of the hand was investigated.

[3] Demonstrated to Neurological Society, June, 1905. Also see "Boyle Lecture," 1905.

Case and Lesion found at Operation[1] or at Autopsy.	Abstract of Sensory Disturbance and Errors of Localisation.
Case 3.—J. C., male, aged 56 years [Sir Victor Horsley]. *Operation*, October 21, 1905. Small myxoma about 3 cm. in diameter situated in middle third of ascending parietal gyrus. There was a considerable amount of softening in the corona radiata beneath the cortex, which certainly involved the fibres of the ascending frontal as well as the ascending parietal gyrus and probably also the fibres of the paracentral lobule.	(*A*) *Before operation.*—Hemiparesis of upper limb. Definite atopognosis proximal in right hand. (*B*) *Twenty-four days after operation.*—Anæsthesia to light cotton wool touches all over dorsum of all digits, pain and temperature sense normal. The topognostic error was pre-axial on the dorsum of the digits as well as proximal. The latter error occurred as far as the carpus.
Case 4.—G. P., male, aged 62 years [Sir Victor Horsley]. *Operation*, June 17, 1905. Small glioma occupying the breadth of the right ascending frontal gyrus and extending to the anterior wall of the fissure of Rolando. The corona radiata, though compressed slightly, was nowhere softened. The lesion made by the extirpation of the tumour involved the anterior third of the width of the ascending parietal gyrus opposite the crater in the ascending frontal gyrus.	(*A*) *Before operation.*—Marked atopognosis in the left hand (dorsum of digits). (*B*) *After operation.*—Marked proximal atopognostic error of all digits of left hand. Very marked post-axial error of the same digits.
Case 5.—W. D., male, 22 years [Dr. Aldren Turner]. Pulmonary tuberculosis. *Autopsy* (*a*) Small tubercle ¾ cm. in diameter growing from pia at junction of posterior part of second frontal, with ascending frontal in left hemisphere. (*b*) Similar tubercle 1½ cm. diameter in mid third of post-cerebral convolution on left hemisphere, and another of about same size at extreme upper limit of post-cerebral convolution. A fourth in right hemisphere ¾ cm. in diameter in first frontal convolution.	Case of multiple lesions of the kinæsthetic area. Attacks of spasmodic twitchings in the fourth and fifth fingers of right hand, and simultaneously of eyelids. Slight motor paresis of the fourth and fifth digits. Slight anæsthesia to light touches on the fourth and fifth digits. Error of topognosis very slightly distal and pre-axial on the fourth and fifth digits.

[1] We do not, of course, desire to claim that the estimated position of a lesion exposed in an operation is necessarily correct, but offer the facts according to our observation at the time.

It thus appears that in each of these unselected cases of what must be regarded as marked lesions of the kinæsthetic cortex there was a topognostic error of response to light touches in two directions at right angles to each other. It is comparatively easy, now that the fact has been definitely ascertained, to suggest from *a priori* reasoning that this biaxial error must exist if the destructive effect of a lesion is considerable, for after all the topognostic sense is estimated by the conscious appreciation by tactility of a spot in a plane, namely, that tangential to the point stimulated. In all such cases, therefore, the error would not only occur proximally by segments along one axis, but would be discoverable in both the principal axes (at right angles to each other) of that plane.

We have now to consider the meaning of the fact that as the above table shows the error in the vertical axis, *i.e.*, parallel to the central axis of the limb, is as usual proximal and segmental, and that in addition there is an error in an axis at right angles to the first which is post-axial or pre-axial in type. Further, this second type of error necessarily bears a relation to the mesial sagittal plane of the whole body, and, moreover, increases as the investigated hand is moved away and placed in successive sagittal planes distant from the mesial plane. This observation in fact raises the question whether with our erect posture the type of sensory representation in the spinal cord is not more relative to the mesial sagittal plane of the body than to the cephalic and caudal sides of the limb axes as in quadrupedal animals. It is, we suggest, at any rate now demonstrated that in a case of lesion of the kinæsthetic cortex, the spinal type of sensory representation, *i.e.*, one parallel to the principal axis of the limb, asserts itself in all compound movements and in conscious perception of touch and points of contact in space. Hence in all future clinical investigation of cases of cerebral anæsthesia changes in axial as well as segmental representation must be looked for.

We cannot, however, discuss this point as fully as we wish, because the normal error of orientation in space of a spot on the surface of the skin is only even now being

investigated by Dr. Slinger, and we have, therefore, no data yet for absolute comparisons.

The foregoing conclusions enable us to suggest an explanation of a fairly common clinical condition, which apparently has not attracted much attention, but which we consider is a further proof that the principal axes of the limbs have a differential localisation in the pallium.

The facts are as follows:—

(*b*) *Representation in the Cerebral Pallium of the Mid Axis of the Upper Limb.*

In the study of sensory disturbances due to lesions of a metameric character in the spinal cord or nerve roots, the mid-axis of a limb, and the mid-ventral and mid-dorsal lines on the front and back surfaces of the limb respectively, have always obtained anatomical attention. The eighth cervical nerve area supplies these central lines or regions of the forearm and of the hand, including the middle finger, and to a less degree the ring finger, with perhaps a slight overlap on each side of the total area. One of us has during many years occasionally observed cases of tumours involving the kinæsthetic areas in which the fits of Jacksonian epilepsy have commenced with a definite sensory aura in the two middle fingers of the hand, then passing up the forearm in its mesial axis. Although we are not in a position to offer precise information on the localisation of the lesion causing this mid-axial phenomenon, still it is such an obvious corollary to the pre-axial and post-axial representation we have just discussed, that we bring forward the following case as a good example of the clinical fact:—

Cerebral Tumour with Sensory Fits Commencing in Middle and Ring Fingers.

Case and Lesion found at Operation or at Autopsy.	Abstract of Sensory Disturbance and Errors of Localisation.
Case 7, T. A., male, aged 47 years [Dr. James Taylor]. *Operation*, November 30, 1905. Large subcortical tumour lying beneath the right ascending parietal convolution, spreading forward to Island of Reil and backward to supra-marginal gyrus.	(*A*) *Before operation.*—Sensory fits, *i.e.*, aura only commencing with sensation of pins and needles in third and fourth fingers of left hand, spreading to arm, face, trunk and leg of same side—followed some weeks later by Jacksonian fits starting in left side of mouth, and spreading to arm and leg of same side. (*B*) *Seventy-three days after operation.*—Dorsum of left hand anæsthetic to cotton-wool. Medium pressure with pin point on dorsum of ungual phalanx showed besides proximal error, reference towards mid-axis as follows:—

Dorsum of Ungual Phalanx.	Digit Stimulated (2 obs.).		Point indicated by patient with Left Index.
	Thumb	1 ..	Proximal phalanx middle finger
		2 ..	Middle phalanx index finger
	Index	1 ..	Distal phalanx thumb.
		2 ..	Proximal phalanx index finger.
	Middle	1 ..	Middle phalanx index finger.
		2 ..	Proximal phalanx middle finger.
	Ring	1 ..	Proximal inter-phalangeal middle finger joint.
		2 ..	Proximal inter-phalangeal middle finger joint.
	Little	1* ..	Proximal inter-phalangeal middle finger joint.
		2 ..	Proximal inter-phalangeal little finger joint.

* Stated by patient to be felt by him on the middle finger.

It is therefore, we submit, perfectly clear that what may properly be termed the mid-axial line and region of the hand and forearm, is as definitely represented in the cerebral pallium as it distinctly is in the spinal cord. It is, perhaps, natural to expect that this should be so, because any contrast between the post-axial and pre-axial portions of the limb respectively, presupposes an imaginary frontier line between them, and, further, the cerebral memorialisation of the transfer of the limb to and from the mesial plane of the body demands that the cerebral pallium should have particularly localised within it the orientation of the middle point of the limb which is thus translated from or to the sagittal mesial plane. This further proof of similarity between the sensory representation in the spinal cord and cerebral pallium is confirmatory of the other phenomena we have described.

We cannot conclude this note on the spinal type of sensory representation being re-represented in the pallium without referring to the very remarkable case published by Sir T. Grainger Stewart [18] in 1894, which illustrates in particular the localisation of the mesial aspect of the limb.

In that unique case there was a disturbance of normal sensation over the whole limb excepting a narrow strip on the ventral and dorsal surfaces respectively. This strip followed the general line of the eighth cervical area and terminated in the middle digit. It was in fact a demon-

stration of the mid-line or axis region. So too the confusion in the patient's mind between the pre-axial and post-axial representation showed that some (functional) disassociation had occurred leaving the frontier line between the two fields of sensation alone unimpaired.

Though the case in question, one of exophthalmic goitre, appeared to be partly a functional one, there can be no doubt as to its value in helping to establish the suggestion we now put forward that the spinal representation of tactility finds an echo in the arrangement of that function in the sensory cerebral cortex.

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THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

At a Pathological Meeting held at 11, Chandos Street, on Thursday, March 1, at 8.30 p.m., the following communications were made:—

Dr. L. S. Dudgeon.—*Acute diphtheritic toxæmia with special reference to acute cardiac failure.*

An investigation was made of various portions of the bodies of fifteen patients who died within fourteen days from the onset of the attack of diphtheria, of fifteen control cases and also of large numbers of rabbits and guinea pigs. The extra-cellular toxins of the diphtheria bacillus were obtained from the experimental work. The heart muscle was carefully studied in every instance and the microscopical appearances were compared with those which occurred in other parts of the body. Dr. Leathes estimated the total quantity of fat present in the poisoned hearts of six rabbits, and compared the results with those obtained from the hearts of six normal rabbits. The microscopical appearances of fatty change produced by the action of the diphtheria toxins formed the main subject of this enquiry.

Lantern demonstration by Dr. Gordon Holmes and Mr. J. H. Parsons.—*The pathological anatomy of amaurotic family idiocy.*

The brains of the two patients examined were of good size, but their convolutions were small and appeared wasted in some regions. There was no macroscopical evidence of defective development.

All the cells of every part of the central nervous system as well as those of the dorsal root ganglia had undergone considerable change. They were swollen, often very much enlarged, their nuclei generally lay excentrically and no tigroid masses were, as a rule, visible in the greater part of the cell. Fine granular material around the nucleus was in many cells the only portion which stained with the basic aniline dyes. The protoplasm of some cells was in addition vacuolated. Varicose enlargements of the dendrites were often visible.

The neuro-fibrils (studied by Bielschowsky's method) were, as a rule, unaltered in the cell processes, but were generally broken up in the centre of the cells. Curious bladder-like processes were attached to many of the cortical cells.

There was some loss of myelinated fibres in the cerebral cortex, especially in the tangential layer, and in the corona radiata in all lobes of the brain, but it was not great. In the brain-stem the cortico-pontine tracts had suffered most severely and there was considerable atrophy and degeneration of the pyramidal tracts. The dorsal columns of the spinal cord were normal, but there was some diminution of fibres in the ventro-lateral columns, greatest in the areas of the pyramidal tracts. The optic tracts and optic radiations were practically normal.

Marchi's method revealed diffuse degeneration in all parts of the central nervous system, but it appeared to be only slight.

There was considerable increase of neuroglia, but this proliferation has only the character of a secondary process and was limited to the areas of the degenerated tracts.

The meninges and blood- and lymph-vessels of the central nervous system were normal and no evidence of old or recent inflammation was visible.

No pathological alterations were found in the peripheral nerves or skeletal muscles which were examined.

The following conclusions may be drawn from the microscopical examination :—

(1) That it is a primary disease of the nervous elements ; (*a*) the neuroglial changes appear to be only secondary ; (*b*) there is no evidence of any vascular disease to which the changes might be due, and nothing to indicate an inflammatory process.

(2) That it is primarily a disease of the nerve-cells and to which the affection of the fibres is only secondary.

(3) That, as the neuro-fibrils are relatively less altered than the interfibrillar protoplasm, the affection of the latter is probably the primary change.

(4) That it is not a condition of arrested development.